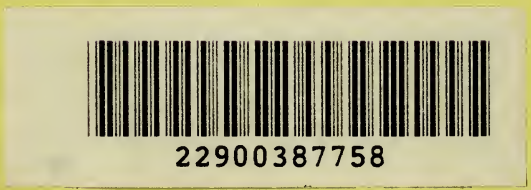
Lectures on Medicine.

By

Samuel Wilks, M.D., F.R.S.



LECTURES

ON

THE SPECIFIC FEVERS

AND

DISEASES OF THE CHEST

BY

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INTRODUCTION

Before commencing the course, let me reiterate the precept which is constantly being forced upon your attention—that lectures cannot afford you directly the knowledge which you require, but are only the instruments to be made use of in acquiring it. Let me explain. I for convenience' sake take diseases *seriatim*, and describe to you the symptoms which belong to them, but the knowledge which I make use of for myself is of quite a different kind; this I cannot directly impart to you. What I am doing in the practice of my profession, and which you likewise will have to follow, is to recognise symptoms, put them together, and draw the inferences from them as to the existence or not of a particular disease before you. Now I should like to teach you how to accomplish this, but the attempt being altogether impracticable, the only mode left is to take the system as found in your books, and given in your lectures, and ask you to do your best with it. You see that the true or practical process is an analytical one, whilst the one we teach is of a directly opposite character; you therefore have to take our system, and invert it before it can be of any use to you. For example, of what advantage is it to you to know the symptoms of pneumonia unless you are able to recognise these symptoms in a patient, and draw from them the right inference as to the existence of the disease. The information you acquire from books and lectures is only a kind of ladder to lead you to knowledge of a higher kind, and when you have reached this you may discard the method by which you have obtained it. When you have passed your examination you will never again have to repeat the symptoms of pneumonia, but you

will every week be forced to interpret the symptoms which lead you to recognise the complaint. It cannot be expected that students can gain much of this practical knowledge until they begin to require it, and therefore it is that they generally fail in answering any but book questions at examinations; for instance, if a candidate be asked how he would set about discovering what was the matter with a patient who had been recently taken ill with oppression of breathing, instead of giving the differential diagnosis of chest disease, he would say the patient had pneumonia or bronchitis, and then set about describing those complaints; but you can perceive how inferior this kind of knowledge is.

If you remembered every word I have said in lectures or every line you had read in medical books, the information is of no use until you have digested it, assimilated it, and then appropriated it to its right use. If you knew every word which had ever been written about pneumonia, you would still be as at great a loss to recognise the disease as you would be in discovering arsenic in an organic fluid if you had only attended lectures in which the properties of arsenic had been described to you.

Another point to which I wish to draw your attention is the subject of *diagnosis*. Some of you probably suppose that symptoms are pathognomonic, that is, that they point unfailingly to certain morbid conditions. This is, however, very rarely the case. The following is the mode by which we suspect the existence of a disease, whether we adopt it consciously or unconsciously. We endeavour to acquaint ourselves with all the diseases to which the human body is liable by observing the sick in the wards of the hospital, and by examinations after death; and we then see what symptoms are associated with particular morbid states. The symptoms themselves usually only imply that certain physical changes have taken place, but the causes and character of these changes can only be inferred from a number of circumstances, from which a high probability is drawn in favour of a particular disease. For example, a patient comes before us with a history of attenuation, cough, spitting of blood and phlegm, and on examination we find crepitation over one apex, and we form a diagnosis of phthisis. We do so because we are acting on a previous knowledge that there is a disease called phthisis commencing at the upper part of the lung and attended by the symptoms just named; but a medical man who was unacquainted with the history of the case, and who being blindfolded did not know at what spot his stethoscope was placed, could not diagnose

the disease from the auscultatory sound alone. We once had here a remarkable case which was believed to be pleuro pneumothorax, but which really was an instance where the stomach had ruptured into a large circumscribed space below the diaphragm, and so gave rise to every sign of pneumothorax. The physicial conditions were exactly the same as in this disease, clearly showing that the final diagnosis of pneumothorax rests only on a very high probability. Tumours in the chest and aneurisms are constantly confounded because their effect is alike in compression of the surrounding parts; thus leaving the final diagnosis of one or the other to probability gained by a consideration of several other circumstances. When, therefore, you are in the wards and the physician makes a diagnosis, you should ask him the reason for his conclusion, and he will say that the symptoms and signs prove a derangement or disease of a certain function or organ, and that his experience leads him to think this is due to a particular cause. The student will often make out the first part of the diagnosis as well as his master, but he fails in the second part from want of experience.

Now, a third remark about *treatment*. I shall do no more than tell you what kind of treatment has been found most successful in the hands of the profession and in my own, and ask you to adopt it. I wish the time had arrived for the adoption of a scientific method, so that I could teach you the therapeutic art according to given rules. It would be far more satisfactory to you could the lecturer on medicine tell you what you are to do under such and such circumstances, or in other words how you are to treat certain symptoms. This cannot be done, for we do not yet know the meaning of many symptoms, and therefore whether or not they should be interfered with, nor do we know the operation of medicines upon them. Let me explain by taking so common a complaint as acute rheumatism, and telling you that the profession is not yet agreed whether excessive sweating is a favorable or unfavorable symptom; therefore we have no right with our imperfect information to try and check it or increase it; we do not know, moreover, in this disease whether the patient has less or greater chance of internal complications according as the arthritic inflammation continues or is speedily arrested; or take typhoid fever, and we find our forefathers attempting to stop a diarrhœa which many at the present day believe to be eliminative, and also endeavouring to promote the feverish process which they thought to be nature's method of throwing off the disease. At the present time we are beginning to think that a reduction of

the fever-heat by external application of cold is efficacious. You see, therefore, with imperfect knowledge the only safe rule is the one founded on experience, and to place the patient under those circumstances of regimen and medicines which you have found most useful.

Then as regards remedies, these are named from their actions on the healthy subject, but their use varies much according to the morbid state of the patient. You cannot say, for example, that a medicine is a tonic, and therefore give it to every one who has a weak pulse, for one weak pulse is benefited by iron or quinine, another by digitalis, another by opium, and another by alcohol. How often have I seen a patient grow weaker by alcohol, and his pulse quickly restored by digitalis, because he had a feeble and dilated heart, and another immensely improved by opium, when every tonic failed, because he had diabetes. Again, I have seen a case of obstinate sickness fail to be arrested by all the usual remedies for this condition, but speedily allayed by iodide of potassium, because the symptom was due to a syphilitic gumma on the brain. The crude belief that every weak pulse is to be remedied by alcohol, because certain forms of weak pulse may be benefited by it, has given rise to the prevalent treatment of all diseases by alcohol. I was once constantly meeting a medical man in this neighbourhood who declared that he gave all his patients alcoholic stimulants because they were all weak; he might as well have ordered them all quinine or digitalis. The method I teach you is this: try and find out the cause of the disease or the symptoms, and then ask yourself what is the remedy which the experience of the profession has found to be the most serviceable in that complaint.

FEVER

Medical diseases may be divided into two great classes—those which originate in the body under the influence of the ordinary circumstances surrounding it, and those which arise from specific external causes. The former are for the most part slow in their growth, or, if they occur suddenly, very often owe their origin to some predisposing condition of the patient. The latter, on the contrary, occur in all alike, irrespective of age and temperament, but have a special tendency to attack the young and healthy. Thus, children are especially prone to the exanthemata, and the natives of newly-discovered countries have ever been ready to fall a prey to the contagion introduced from the civilised world.

As I am about to commence with the specific diseases, in nearly all of which a febrile condition exists, I shall make some preliminary remarks respecting fever in general.

Fever or feverishness accompanies every local inflammation, and nearly every specific or zymotic disease; there is, therefore, no reason why any one class of diseases should be selected for the designation "fever." The word was adopted in ignorance, and probably for the following reason:—If feverishness was attendant, for example, upon pneumonia or other local inflammation, the latter was sufficiently well marked to suggest the appropriate name for the disease; so, also, if the feverishness was accompanied by such peculiar symptoms as a pustular or red rash on the skin, these rashes would be sufficiently striking to suggest such names as small-pox or scarlatina; but if with a similar constitutional disturbance there were no markedly distinctive symptoms present, the general feverish condition would alone have attracted attention, and therefore given a name to the disease. It consequently happened that after our forefathers had named diseases after any characteristic features which they might possess, they were fain to call others attended by

febrile disturbance "fevers," and which we now know include several kinds of complaint. At the present time, therefore, we should endeavour to expunge the word "fever" as indicative of any particular class of disorders, especially as the disease best known by the name—"typhoid"—is wanting in many of the characteristics which are supposed more especially to attach to fever. We should have no difficulty in getting rid of the term were it not for the two adjective expressions — typhoid and relapsing — and which require the word fever to be added to them. In the first we might substitute "enterica," but the relapsing fever is still waiting for a name.

Now, as regards fever *per se*, or feverishness. This belongs essentially to local inflammations and specific diseases, so that if you are called to a patient who has been for a day or two in a feverish state the probabilities are greatly in favour of his having either one of these conditions. If, after careful examination of the body, no local inflammation can be found, then you must think of a specific disease, and endeavour to determine its nature by the history of the illness and the presence of characteristic symptoms, such as rashes, &c. If there be none present, the disease is usually called typhoid, because this is a disease in which the characteristics are frequently wanting. It is for the same reason that if a local complaint is overlooked the case is designated by the name typhoid fever. I will now ask you to add to the category of local inflammation the case of tuberculosis, in which feverishness or pyrexia also occurs, and to add to the second category of specific blood diseases the case of pyæmia or septicæmia; you will now probably have very nearly all the conditions under which fever exists.

When I say that the feverish state implies in nearly every case one or other of the above-named conditions, it must be remembered that in certain lesions of the nervous system the temperature will be considerably raised, but I could not maintain that this increment of heat is equivalent to fever.

The feverish state or pyrexia is marked by shivering, preternatural heat, quickened pulse, hurried respiration, furred tongue, usually dryness of skin, loss of appetite, pain in the head and limbs, dark-coloured urine, and indisposition to any mental or bodily exertion. Some of these symptoms may be absent, but the feeling of tiredness or irksomeness never. The most important or constant element in pyrexia is increase of heat; but it is not absolutely a sign of fever, for, as just now said, it may occur under various lesions of the

nervous system, and on the other hand we sometimes find in the course of a local inflammation where the other symptoms of pyrexia remain, that the temperature has been reduced to the normal standard.

The temperature of the body is 98·4° Fahrenheit, as we find it in the axilla. In the mouth or rectum it is higher, and in the inside of the body it is 100° or more. This precise and uniform temperature in the human body is a very striking fact; it seems as if the integrity of the frame depended upon it, and that any departure is attended by some morbid condition or ill-health. The human body is a wonderful machine or furnace, perpetually burning, and maintaining itself at a temperature of 30° or 40° above that of surrounding inert objects. This production of heat is generally agreed to be mainly due to a chemical process, about which you have heard all particulars in the Physiological Lectures. Not only is there combustion going on in the lungs, as formerly thought, but analogous changes in all the tissues of the body; in every gland heat is evolved, and most of all in the muscles. If too much heat is produced it is given off by the skin; if the loss should be too great we cover ourselves with clothes so as to retain it. It is therefore evident that man could live only in a state of nature in some favoured regions of the globe. The heat is regulated by natural and artificial means, and the standard is the balance struck between heat produced and heat lost.

You might be reminded of Sir B. Brodie's experiments of decapitating an animal and keeping up artificial respiration, followed by a lowering of the temperature, which led him to believe that the nervous system was essentially active in the production of heat, and that the chemical process would not alone account for the amount. I mention this because it is known that the temperature is often raised in nervous disorders, but whether it be due to the influence of the nerves over the blood-vessels, and so upon nutrition, or whether nerve force is converted into heat in a more direct manner is a *quæstio vexata*.

It is no business of mine to enter upon this physiological question. and I only do so because it is necessary for the true elucidation of "fever." Does fever imply an excess of those processes which are constantly going forward in health? The question includes the generally received opinion, and if true my task is comparatively easy. I have merely to demand of you a recollection of what you have been taught elsewhere, and apply it to the subject under review.

You have to think of the causes which are in operation for the production of animal heat, and then consider that all these are acting in an excessive degree in the case of fever. Although there are good reasons for this conclusion, yet it is not entirely free from objection, for if a person takes violent exercise, and his temperature is raised, respiration more hurried, pulse more rapid, more solid matter in urine, and, in popular language, is "all in a fever," nevertheless the tongue is not furred, nor is there anorexia as in true fever. It may be that the latter symptoms would be induced did the temperature keep high for any length of time, but of this there is no proof. It has been thought that fever may be due, not so much to increased production of heat as to a diminished loss from the skin; but in answer to this I may say that the result of experimental inquiry shows that more heat is given off in fever, and with a corresponding tissue change throughout the body. I might also allude to other theories, such as that propounded by Dr Maclaghan, at the last meeting of the British Medical Association— that there is certainly an increased chemical change in the fever state, but in the case of the specific diseases this takes place by the "poison germs" or "contagion" acting, not on the made tissues of the body, but on the circulating albumen derived from the food; there is truly an increased combustion process, and the body wastes, but this is because it is starved of its proper nutriment.

It is very interesting to compare the chemical change and heat product with those of health, for we thus gain a notion of the amount of wear and tear which is going on during a severe illness of the fever kind.

Professor Houghton has calculated that if you look at a man lying on his bed—the subject of typhus fever—you have the counterpart of a traveller who has been performing a fifty-mile journey in twenty-four hours as regards the amount of heat produced and tissue change which has taken place in both of them during that period. You may also make a calculation of this kind: Say a man, weighing 150 lbs., has walked up Mont Blanc, a height of 15,000 ft., which is the same thing as that weight having been carried up. Now, if you refer to your syllabus of the Natural Philosophy Lectures you will see the meaning of the unit of mechanical work and the unit of heat; also that the mechanical equivalent of heat is 772 foot-pounds, that is, that a force required to raise a body one degree of heat would lift it 772 ft. Now, if you divide 15,000 by 772 you get nearly 20, so that the force required to take a man up Mont Blanc,

would raise his temperature 20°. Suppose, therefore, that we had a patient whose temperature was raised in a short time to 10°, the same amount of force would have been expended as if he had walked half way up Mont Blanc. Or if during the day it had reached fever height or 6° above normal, the same amount of tissue change would have occurred in the patient as if he had ascended Ben Nevis, the highest mountain in Great Britain, for it approaches 4632 ft., the calculated distance. The amount of chemical change due to combustion could be reckoned from this, and the amount of fuel required for the process. In this way calculations may be made of the greatest scientific interest and value.

You may ask whether the increment of heat in fever has been proved to be proportionate to tissue change by the increased amount of excreta voided by the body? This has not been accurately ascertained, as the investigation is a difficult one, for it would be necessary to analyse the secretions from the intestine and from the surface of the body, as well as the urine, and the expired air from the lungs. As regards the urine, observers differ, but it is generally admitted that the solids are increased; the urea, uric acid, phosphates and sulphates, but not the chlorides which are deficient in febrile complaints. The increased amount of carbonic acid given off by the lungs is two and a half times more than in health, and therefore this alone would go far to account for the increased tissue change and corresponding increment of heat.

No fact connected with the use of the thermometer has been of more interest, or perhaps of more practical value, than that which associates a necessary stoppage of vital action with a great departure from the normal standard of heat. Thus, the normal temperature being a little more than 98° F., fever heat has an average of 104°, and there is good reason for the belief that this excessive heat to which the animal body is raised is productive of many of the symptoms and ill consequences which are observed in the febrile state. We are no longer satisfied with regarding this state as a necessary accompaniment of a local inflammation or specific disease which must run its course, but that the high temperature may be fruitful of further danger, and should, therefore, be especially attacked by refrigerant means. A temperature of 109° or 110° is incompatible with life, and thus we have at last found a clue to the cause of death in sunstroke, and in some remarkable disorders. The former, which is more properly called heat-stroke, may occur in a hot climate in the night as well as the day, and is due to the

sudden rising of the temperature of the body from the external heat, and inability to throw off the retained heat from some peculiar state of the atmosphere. If the temperature of the body rises 10° or 12° above normal, the blood and muscles undergo a change, and life must cease. In one of the late summers a lad wheeling a barrow down Fleet Street was taken suddenly ill and carried to Bartholomew's Hospital, where he died in an hour. His temperature was found to be 110°.

It was not until lately that we were able to explain the phenomena attending a very fatal form of rheumatic fever. There had long been known a complication styled metastasis to the brain, in which the arthritic inflammation subsiding, the patient became delirious and quickly died, the post-mortem examination showing no visceral change to account for death. We now find that at the onset of these symptoms the temperature quickly rises until it may reach 108° or 109°, and in a short time the patient dies. This condition is called one of hyperpyrexia. With this important fact the appropriate treatment is obvious—to reduce the heat by any possible and speedy method. The house is burning, and the fire must be extinguished. The patient must be put into a cold bath again and again until the heat is reduced, and the immediate danger has passed. The plan has been adopted in many cases, and with the most successful results. In the same manner the cold bath is being used in typhoid and other febrile states when the temperature rises to a dangerous height. It is said that throughout many parts of Germany the mortality in typhoid fever has been much reduced since the adoption of the cold-water treatment.

Whether, when death has occurred, combustion can still go on until the body is consumed, I do not know. Some writers have spoken most positively as to undoubted cases of it, whilst the profession generally remains incredulous. Some years ago a controversy took place between Mr Dickens and the newspaper press on the subject of spontaneous combustion, in consequence of the author stating that when Mr Krooks' room was entered nothing was found in his chair but a little grease and a cinder.

What is the lowest temperature which can support life I cannot tell you, but that all vital action is suspended when the heat of the body is much lowered is a self-evident proposition. If this is so, it is no doubt true that all departures below the natural standard are injurious to the normal processes of nutrition which are constantly going on in the body. Sufficient attention has not been paid to the

subject, but at present I will merely draw your attention to the fact that all the wonderfnl processes which are constantly in operation in the interior of our systems require a temperature of 100°, and that the fœtus was developed during the nine months it was inside its mother's womb at the same temperaturc. I will also draw your attention to the opposite fact of a low temperature being instrumental in arresting every stage of development and growth, as is seen in the fact of the possibility of carrying the ova of fish to the Antipodes if preserved in an ice-cold chest. A practical lesson might be learned in asking you to consider well these facts when you have to treat wounds on the surface of the body. It is no business of mine to advise you as to your procedure after surgical operations, but I cannot be doing wrong when I say that for the healing of a wound a certain degree of heat is required, and that if this be equal to the normal heat of the body, the nutritive processes will proceed the more readily. I need not say what my reflections are on this subject when I hear so much of the cleaning of wounds to get rid of putrid secretions and imaginary poisonous germs, and for this purpose am told of the stump of an amputated leg being exposed to a constant stream of cold water, by which its temperature is reduced to 50° or 60°, and when I at the same time know that if all the fingers of the hand are crushed and immediately bandaged up, perhaps in a clumsy manner, yet in a few days the severed parts will grow together again with an extraordinary rapidity. Healing by the scabbing process is also very rapid, both in man and the lower animals. Think, therefore, again of health and healthy processes in connection with a remarkably fixed temperature. We are indebted mainly to Wunderlich for teaching us the importance of the rise and variations of temperature in the human body as indicative of unhealthy states. He has endeavoured to show that in disorders attended by febrile disturbance the rise of the temperature will at once point to its approach, and according as this rise is sudden or slow, so may the various forms of specific and local disease be anticipated. It is, no doubt, true that the rise varies in different disorders, and could I thoroughly rely upon the statement of Wunderlich, I would compare his tables of temperature so as to enable you to gain at a glance an idea of their differences for the purposes of diagnosis. Later observations, however, have shown that these are not altogether trustworthy, and therefore, until we have arrived at more accurate conclusions, I think I had better not endeavour to lay down any rules respecting the progress and

decrease of fever in these complaints. I shall wait until I treat individual diseases, and then tell you what we positively know from observations of our own in respect to this subject.

When we make a table of temperature we speak of the increment of heat as the "rise," and the fall as the "defervescence;" when the latter is sudden it has been styled the "crisis," and if gradual the "lysis." It has been said that with each degree of heat the pulse rises 10 beats. This is not absolutely true, but a proportion of the kind does exist. For example, with a temperature of 98° and a pulse of 60, with a temperature of 99° there would be a pulse of 70, and with a temperature of 104° a pulse of 120.

SPECIFIC OR ZYMOTIC DISEASES

Before taking each of these separately, I shall first speak of them as a whole, and you will then perceive what features they have in common. The most approved theory is that each owes its origin to a specific or peculiar virus, which being introduced into the system propagates therein, and reproduces itself to an almost infinite extent. This process is attended by certain characteristic phenomena, and, as a rule, the subject of it cannot undergo the process a second time. These facts suggested to Liebig the idea of a fermentation in the blood, whereby a certain material (like sugar or starch) might undergo a fermentative change, and that when the process was ended with the destruction of the agent it could never occur again. Upon this hypothesis he framed the word "zymotic" (*zumoo*, I ferment), a term still retained, although the idea attached to it is not generally held, but used synonymously with the expression "epidemic." An explanation by others of a non-recurrence of a specific disease is found, not in the idea of fermentation, but of growth, whereby the virus introduced into the blood grows at the expense of a certain material within it. These two theories, however, may be identical if the growth of the yeast plant and fermentation are in any way allied.

We use the term "specific" to designate these diseases, because we believe each one stands isolated from the others, in the same way as a plant or animal is distinguished from all others, as having a different parentage. Just as in the organic world every example of tree or beast produces its like and no other; so, amongst these diseases, small-pox produces small-pox, scarlatina produces scarlatina, and none other. Then there is the absolute proof that if the virus of one of these diseases be inoculated into a healthy person a similar affection is produced in the latter. We have, therefore, every reason to conclude that whenever we meet with an example of a specific disease that the germ of the disease has been implanted in the subject of it

just as when we see a weed in our garden we believe the seed must have been sown. There are also many other arguments in favour of the belief of the specificity of these diseases, and more especially those drawn from their history in different countries. As, for example, although the climate is the same as it has ever been, yet it was only three years ago that scarlatina first appeared in India and parts of Australasia after the arrival of a ship with cases of the disease on board. Small-pox was unknown in America until the conquest of Mexico, when, on being introduced, millions fell victims to its virulence, and the same with syphilis in the Pacific Islands. The importance of these questions arises from the fact that it is only in the minority of instances of specific diseases that we can trace contagion, and yet I must advise you to consider that in every case the disease has been "caught." In the present state of our knowledge you have a right to assume this, even though you are unable to prove it, and to let the *onus probandi* lie on those who maintain the contrary, or the spontaneous generation of these diseases.

You can understand why the specific cause of these diseases is spoken of as a poison, since in the animal and vegetable world a poison means that which produces certain definite and distinctive injurious consequences; so peculiar, that when meeting with these results in any individual case, we immediately recognise the causes which have set them in operation. If, for example, a small quantity of arsenic be administered to a human being, he is immediately seized with vomiting, purging, collapse, and very definite symptoms; and in the same way, if he swallowed the cholera poison or contagium, very similar phenomena would ensue.

It is, therefore, held that each disease has its cause or active principle, which is called the "contagium," and the time may come when a future lecturer may place before his class several bottles labelled "vaccine," "varioline," "typhine," "cholerine," &c. That this contagium is not merely a homogeneous fluid, but contains distinct germs, is proved by diluting it, when it is found that after many experimental trials at inoculation a certain number of these only are successful, showing that in these cases the germs are accidentally caught. It is generally believed that these germs are living particles or "zoosperms," and that they can only grow in the animal organism; therefore it is possible that all the zymotic disorders might be stamped out by isolating patients, just as the cattle plague is stamped out by killing the animals who are the subject of it.

There are those, however, who, holding to the specific nature of the "contagium," believe rather that it is of a vegetable kind, and therefore can have an altogether independent existence outside of the animal body. They contend that the poison of cholera is a rice fungus, that of measles the same fungus as a well-known mould, that typhus and typhoid are due to fungi to which Hallier has given distinct names. There are those who, holding to the specificity of these diseases yet believe that they can be spontaneously generated; that typhus fever, for example, is produced by overcrowding, relapsing fever by impoverishment, typhoid by vegetable decomposition, cholera by an exaggeration of the circumstances present in our autumn months, scarlatina by decomposition of blood, and even small-pox (according to Miss Nightingale) by bad sanitary conditions. Syphilis would be said also to arise *de novo* from promiscuous intercourse.

I give you these opinions, not with the object of forcing any one of them dogmatically upon you, but that you may know what the theories are, and that you may reason logically and clearly upon them; that you may not raise a cry against the dirty river in your town whenever scarlatina, typhoid or cholera appear, unless you can explain in what way you hold it to be guilty of their production. At present the prevalent opinion would be that the river merely acts as a conveyer of distinst and separate poisons at different times. The proof apparently lies in such an example as is given by the Medical Officer of the Privy Council. A notoriously dirty and ill-drained village, where the wells are frequently contaminated from the offensive ditches, is free for many years from typhoid fever until a case of this disease is brought into the place, when immediately it spreads widely amongst the inhabitants. The means of conveyance is at hand when the poison arrives.

Now let us see what happens when a specific virus is introduced into the system. It immediately infects the blood, and commences to produce some change in the way of altering its constitution, whilst it itself is being infinitely propagated. This is the hatching time or period of "incubation." As a rule the subject of it is not conscious of the change, and therefore is not ill. When a virus is inoculated we see a local inflammation going on, as after inoculation of small-pox, where the pustule is forming eight days before the constitution suffers; or after inoculation of syphilis where an induration goes on at the spot, and after forty days the constitutional symptoms appear. It is most probable that if the virus of other

diseases were inserted directly into the system that the incubating time would be definite, but the mode of introduction differs in so many ways that this period varies considerably in each particular case. It may be due to the mode in which the virus is introduced, the latter being less able to penetrate the system in one direction than another. Thus small-pox virus inoculated affects the system a few days before it would do so if taken in by the lungs as vapour ; so the secretion of measles acts more rapidly than the vaporous emanations of the skin. I must therefore take the extremest and narrowest limits, and give you the average. The importance of this question is brought before you when you are asked, for example, by a mother at what time she is safe from infection after having left nursing her sick child.

When the incubation is complete the whole body becomes affected by being thrown into a violent febrile state, in which every organ of the body suffers. At the same time the surfaces of the body, including the skin without and the mucous membranes within, show marked signs of irritation, and since on these surfaces the poisonous secretions often appear, it is thought that the latter are eliminated by these sources. The alterations on these surfaces are peculiar, and assist us more than by any other symptoms in identifying the disease ; as, for example, the peculiar rashes of the exanthemata, and the affections of the ileum in enteric fever. All the solid organs likewise are affected by the poisoned blood, as indicated by their deranged functions seen in the delirium, sickness, diarrhœa, yellow skin, or albuminous urine. The organs are filled with blood which is often undergoing a solution, and thus hæmorrhage occurs within them as well as on the surfaces of the body. We meet, therefore, with what are called malignant forms of the specific diseases in which this hæmorrhagic tendency is often the principal symptom.

The symptoms are due to the action of the specific poison affecting the blood, paralysing the nervous system, and by setting up the ordinary febrile state by increased tissue-change, poisoning the blood afresh by the products of metamorphosis. The increased heat, too, in all probability may be a cause of some of the more important phenomena.

I would ask you, therefore, to think of these specific diseases as a whole, by which you may remark in what particulars they resemble one another, and you will not be led into error when a new epidemic arises, either amongst men or the lower animals. You will

not assert, because in the rinderpest an eruption is met with, the disease is smallpox; or because ulceration occurs in the intestine that it is typhoid fever; nor will you necessarily think that an epidemic amongst pigs is scarlet fever because the skin is red.

Disease.	Incubation.	Premonitory fever.	Duration of rash.	Mode of contagion.
Typhus .	Average 9 days, few days more or less	4	10	Exhalation from body and lungs, from clothes.
Typhoid .	12—14 days or less	10	14	Secretion from intestine, inhaled or swallowed.
Relapsing	Probably 4—5 days	1	7	Exhalation from body and lungs.
Smallpox	12 days; when inoculated 8 days	2	8	Exhalation from body and lungs, from clothes, purulent secretion and scabs.
Scarlatina	3—6 days or less	1	3	Exhalation from body and lungs, from clothes, blood, epithelial scales swallowed or inhaled.
Measles .	10—15 days; when inoculated 7 days	3	4	Exhalation from body and lungs, from clothes, blood, secretion from nose.
Syphilis .	30 days	Few days	Several weeks.	Blood and various secretions.

I will conclude with a word about treatment. As far as our present knowledge extends there is no cure for specific diseases; they run their course and cannot be cut short. At least, at the present time there is no evidence in proof of their cure, although we are constantly trying every new remedy which is suggested for the purpose, such as belladonna for scarlatina, sulphurous acid for typhoid, or sarracenia for smallpox. Whether, after inoculation on the surface of the body, the constitutional disease could be modified by destroying the focus of contagion at that spot is questionable. I am not aware that any experiments were made in rubbing out the smallpox pustule during its development in the time of inoculation, but it was the opinion of Hunter and some modern surgeons as regards syphilis, that the early treatment of an indurated chancre would modify the secondary symptoms. All we do at present in these specific diseases is to assist Nature, and relieve any especially untoward symptoms. We place the patient in as good air as possible, so as to dilute and carry off the poisonous effluvia, and give him cooling drinks to favour the elimination of the products of tissue change.

At the same time we may give medicines to relieve a bronchitis or check a diarrhœa, and quite recently it has been thought that we may do more than this; for we have begun to regard the increased temperature in the febrile state, not as a necessary part of the process which must be gone through, but as a condition which is positively injurious, and therefore may be checked with advantage. The plan, therefore, of placing our patients with typhoid fever in cold baths is undergoing a trial.

Before speaking of the so-called fevers, I might remind you that the distinction between typhus and typhoid has only been made of late years, and this is the reason why I give them together and contrast them with one another. There was a time when the most conflicting opinions existed in the three capital cities of Great Britain and that of France respecting the nature of fever. In Paris it was maintained that an intestinal affection was the seat of the malady. In Dublin the intestine was pronounced to be healthy, while in London two varieties were described, according as the bowel was or was not affected. The difficulty was cleared up by the demonstration, that under the name "Fever" several maladies were included, such as typhus, typhoid, and perhaps other forms. I shall begin with typhus.

TYPHUS

Typhus has an incubation of nine days. In a carefully observed epidemic some years ago the average time was found to be eight days; in another epidemic the incubation period was reckoned at twelve days, but you may remember nine as the average time. At its termination the febrile symptoms set in violently; there is pain in the limbs, great lassitude or prostration, intense headache, and total loss of appetite; fever heat of 104° is often reached on the second day.

After three days of extreme fever the characteristic symptom appears—the so-called mulberry rash. At first this is bright, and, like other exanthems, fades on pressure; it is indeed like measles, and thus has been styled the measly rash. It is first observed on chest, abdomen, and arms, and then spreads all over the body. The term mulberry denotes an appearance as if mulberry juice had been squeezed on the skin, whereby the whole surface would have been universally discoloured. At the same time the juice by collecting in drops would have formed larger spots intermixed with the more

general staining. If, therefore, you look at a typhus rash you perceive that the skin is uniformly discoloured by a purplish exanthem, and scattered over this are some larger spots. The latter have no definite shape, and are with difficulty isolated by the eye, consequently there is nothing about them characteristic, and the rash in typhus must be judged of as a whole. If the diagnosis cannot be at once made no further investigation will unravel it. This rash lasts ten days; for the first few days it fades on pressure, after that it becomes darker and ceases to fade; the blood stagnates in the capillaries and can no longer be squeezed out. In very bad and fatal cases more than this occurs—some of the blood exudes into the tissues, leaving blackish spots; in this way the body and legs are covered with "petechiæ" and sometimes large bruise-like patches form called "vibices." When this is the case the petechial rash remains after death, whereas every exanthem necessarily fades. I believe children may have typhus without a rash. For the sake of systematic arrangement, you may divide the rash into three stages: 1. The pinkish; 2. the reddish-brown; 3. the livid or petechial.

When on the fourth day the rash appears, the fever with the accompanying symptoms continues, the patient getting necessarily more prostrate day by day; he is drowsy, and deaf, or delirious, the tongue is brownish or reddish at the edges, the respiration is quickened and perhaps there is some little bronchitis, the pulse 120—130, the bowels perhaps confined, and the urine high coloured. All the functions of the body indeed are disturbed. The brain shows the change by all mental power having gone; if the patient be roused to answer he immediately sinks into the same lethargic state again, and speaks thickly and with difficulty. In fact he is in what is often called the "typhoid" state, characterised by extreme muscular and nervous debility. At the end of about fourteen days these symptoms rapidly abate together with the eruption, and convalescence begins. From the height of the fever to its fall from thirty-six to forty-eight hours or less elapse. Thus it may be said that the rise of temperature is rapid as well as the defervescence.

If the case should not end favorably, all the above symptoms become more severe—the rash becomes petechial, as above mentioned. That the prostration is greater is shown by the patient lying on his back sunk down in bed, with his arms often outside the clothes, scarcely sensible and unable to speak, quite incapable of being

roused, with constant muttering, great difficulty in swallowing, constantly picking at the bed-clothes, or at imaginary flies. Pulse very rapid, 140—150, and when felt the muscles are found to be jerking, producing "subsultus tendinum." The patient does not sleep, but lies with his eyes open in a state which is called "coma vigil." The bladder is distended. Bowels act involuntarily, and death soon ends the scene.

With regard to some of these symptoms individually, the paralysed bladder must be especially noted; also the weakened tone of the heart, making the first sound scarcely audible, and if beating more than 140 per minute denoting a condition rarely recovered from. The sinking down in bed indicates the inability of the patient to raise himself up on his pillow. Congestion of lungs is known by the expectoration of mucus tinged with blood; and the polluted air from them may be noted by catching the ammonia on a slip of glass wetted with hydrochloric acid. Occasionally convulsions are observed, and these may occur in connection with albumen in the urine, but also without it. Occasionally swelling of the cervical lymphatic glands. On post-mortem examination nothing characteristic is found; the blood is liquid, saturating all the organs which are at the same time soft. Petechial spots are observed on the skin, as well as on the internal surface of the body, and on the coverings of organs.

Prognosis.—Cæteris paribus, this is according to age; that is, all young persons when placed in favorable circumstances recover, whereas all old persons die. I believe the age of forty would be about that in which the chances of recovery or death are equal, but above this age the risk rapidly increases, and in my own personal experience I never saw a patient above fifty recover. The average mortality of all ages is about 18 per cent.

Causes.—Believed to be specific; but it is remarkable that typhus is only observed amongst the poor when crowded together in the courts and alleys of large cities. Therefore it is not seen amongst the better classes of society, except clergymen and doctors, who of course have caught it in their vocation. These facts have given rise to the belief that the poison of typhus may be self-generated by the production of a new and specific element from the emanations of human bodies, and certainly there is much to be said in favour of this view. For instance, it was formerly known as gaol fever, or ship fever, and was thought to arise whenever dirty people were crowded together in a small space. In the case of a gaol, there

was no proof that the specific poison had not been introduced; but in the case of a ship at sea this could not have occurred; although it is difficult to find good authenticated instances of the disease breaking out at sea and after the ship had been a long time from port. The prison fever was certainly very remarkable from the virulent nature of the poison and the rapid progress of the contagion. Thus we read that at an Old Bailey assize, about a century ago, forty persons were stricken with fever, and several died. Those who were seized were the Lord Mayor, and one of the Judges, several officials and strangers. It states that the windows were open on one side of the Court, and it was those only who caught the breeze, on the other side who were smitten. This shows that the disease is highly contagious, and yet when the patient is in a large room and the air calm there seems reason to believe that the poisonous action ceases at less than a yard from its source. It may be carried in the clothes and probably when the patient dies the typhus poison dies also. Persons rarely have typhus twice.

The *diagnosis and treatment* I shall reserve until I speak of "Typhoid."

TYPHOID OR ENTERIC FEVER

In typhoid fever the most characteristic symptoms have reference to the peculiar changes in the intestines, therefore it may be as well to describe first of all the process which takes place in them. It had been generally thought that the typhoid virus, after entering the system, produces therein certain changes of a fermentative kind, and that the poison having propagated itself in the system to an almost indefinite extent is carried off by the intestinal glands. The incubative period, it will be seen, as well as the whole duration of the disease, is much longer than in the exanthemata, and it is probable that almost from the very time at which the poison enters the body the intestinal glands begin to be affected. These glands are Peyer's and the solitary glands. You know that the former are aggregated in long oval patches, larger and more numerous towards the end of the ileum, smaller and rounder higher up the canal. The spots seen upon them consist of circles of black specks corresponding to the summits of the follicles in the submucous tissue. The solitary glands are of the same composition, and are scattered amongst the Peyer's patches; they are also found in the

cæcum and large intestine. These two sets of glands, at a very early period of enteric fever, are observed to be enlarged and swollen, and, as the disease progresses, become occupied by masses of the soft typhoid material. It is not often that we have an opportunity of seeing the intestines when the process is at its height, but from specimens in our museum this appears to be from the tenth to the twelfth day of the fever, reckoning from the day on which the patient is taken ill. When examined by the microscope the deposit is seen to be composed of cells with several nuclei, but presenting no very characteristic appearance, although they are believed to have a definite and distinctive character of their own. It has long been a question as to the exact seat of the formation, whether the new material is deposited within the flask-shaped follicles or in the intervening tissue. The former opinion was held by most because it could be often shown that after recovery from the disease Peyer's glands had resumed their normal appearance, and that when an evident destruction of surrounding tissues had taken place, it was due to an unusual and abnormal extension of the typhoid process.

The subject has recently been investigated afresh by Dr Klein. He confirms the opinion now held by physiologists that these agminated glands are not excretory, but lymphatic glands spread over the internal surface of the intestine. They are composed like other lymphatic glands of a fibrous reticulum with cell elements, and that in typhoid disease a proliferation of cells takes place until the whole gland with the surrounding tissue is converted into one brownish mass. In the earliest stages of typhoid fever he finds the probable organic germs or micrococci together with mycelium and branched filaments occupying not only the glands but the lymphatic vessels and Lieberkuhn's follicles. The process therefore according to Klein resembles rather what is seen in absorbent inflammation with implication of the neighbouring glands. He does not say whether the virus is carried direct to the intestine or is conveyed through the blood. According to this view an active absorption is going on from the intestinal canal rather than an excretory process.

All that the naked eye sees in enteric fever is a deposit of a peculiar nature, being formed within Peyer's and the solitary glands, with some increase of vascularity around them, and that when the formation has reached its height, a disintegrating and sloughing process is taking place within them. A great part of it may indeed

be again absorbed, whilst the remainder is thrown off in shreds or in complete casts of the oval patches. The typhoid disease is therefore not a simple inflammatory process followed by ulceration, but an altogether peculiar one.

I told you that the affection would reach its height in twelve to fourteen days, and if death occurred at this period you would see these masses of deposit rising above the surface of the mucous membrane, also if two or three met they would nearly close the intestine. After this time they soften and disintegrate, so that if seen during the following few days the surface of the gland appears rough and shaggy; they then become still smaller, consequently at the usual time of death after the third week, Peyer's patches have a rough appearance from portions of the material being still adherent. The solitary glands also present ragged spots about the size of peas. If the tissue intervening between the follicles should be involved, the sloughing process may attack the mucous membrane, the submucous tissue, and all the other structures of the intestine. If sloughing is very active a blood-vessel may be opened, or the muscular and peritoneal coats may slough and a fatal perforation ensue. Or, superadded to the sloughing process, an inflammatory and ulcerative action may take place when a true ulcer may form, which must cicatrise before a cure is complete. It is possible that the true typhoid process which occurs in the glands is little more than what I have mentioned, but if the neighbouring extra-glandular structure become involved, then the various stages of inflammation ensue before cicatrisation is complete.

We will, now, once more commence at the beginning, and suppose that the patient has received into his system the infecting virus or germ. This is slowly producing a change within the body, and probably in a visible manner on the intestine for twelve or fourteen days before he begins to feel ill. The patient often thinks he has taken cold, as he experiences a general malaise with pains in the limbs and other feverish symptoms. During the next few days the patient becomes still more feverish and ill, and as regards the working man whom we admit into hospital, he has often not taken to his bed when he appears on the eighth or tenth day of his illness. It is said, however, by some observers that fever heat is reached on the fourth day; but it is admitted by all that the symptoms approach much more slowly than in typhus, and are much less severe, there being less prostration, less severe headache, &c. Although the symptoms are those which are common to all febrile disorders, there

may be a suspicion of the nature of the impending malady from an early tendency to diarrhœa, or sometimes by an epistaxis, which is a not very uncommon early symptom.

About the tenth day after illness, the characteristic rose rash makes its appearance. The spots are usually observed first on the chest and abdomen, appearing as small points of a pink colour—perhaps half a dozen in number—which in a few hours develope into the true rash. At the same time that these are forming, fresh spots are appearing, and so on from day to day. Each spot lasts four days, and then fades away. There are thus successive crops daily coming forth until the termination of the fever. There may be only ten or a dozen of these spots to be counted at one time, or there may be fifty or more. They may be also observed on the back and limbs. It is only in about four fifths of all the cases of typhoid fever that we meet with them. The designation of Louis, who first described them, will best pourtray their character—"tâche rose lenticulaire," the first word implying a spot which fades on pressure, the second carries its meaning with it, and the third implies a spot which is circular and raised like a lens or watch-glass. It is, therefore, rounded, to be felt by the finger and not acuminated.

If, then, the patient be seen at the height of the disorder, he will be in bed, very ill with all the usual febrile symptoms, but the following points may be specially noted:—First, owing to the chief seat of the disease being in the intestines the main symptoms have reference thereto; thus the abdomen is full and doughy owing to the bowels being distended, and on feeling them a gurgling may sometimes be heard from their contents being composed of air and fluid. There is diarrhœa, the bowels being relieved two or three times a day, the motions being liquid and yellow, or rather as if solid fæcal matter had been powdered up in a liquid; they are thus technically called "ochry," and are said to be like badly made pea-soup. If carefully examined, sloughs of typhoid matter may sometimes be found in them, and they are said to be rich in saline matters, and occasionally blood may be observed to be mixed with the stools. The patient is, as a rule, not so depressed as in typhus fever, his mind is clear, and he may often be observed to have a flush on one cheek; the tongue is very characteristic, the fur having gone; it is left red or beefy, at the same time very dry, glazed on the surface, and often fissured, so that it bleeds when protruded. The pulse is 120—130. The temperature undergoes a remarkable daily rise and fall, and the same occurs at the onset of the disease,

so that it has been said that a sure diagnosis can be made from this sign alone. It is generally said that the temperature falls one degree every morning, and rises again every night, but in several accurate reports taken here the extreme rise was found about four o'clock in the afternoon and then the fall began. Again, it has not been found by all observers that fever heat was reached on the fourth day, but it is more difficult to obtain exact experiments in respect to this. I will give the table of Wunderlich, which you may keep by you for the sake of comparison with any observations of your own :—

	Morning.	*Evening.*
1st day ...	98·6 ...	101·3
2nd day ...	100·22 ...	102·55
3rd day ...	101·06 ...	103·64
4th day ...	102·56 ...	104·44

You will see by this that the rise and fall of temperature exists from the very beginning of the disease, which marks a peculiarity about it. The lungs are frequently much congested in typhoid fever, and therefore a cough with expectoration of a bloody mucous sputum is not uncommon. There is sometimes sickness, and the urine is found as in other febrile diseases. If hæmorrhage occurs from the bowels, it is not to be considered necessarily of grave importance.

The patient lies in this condition until three weeks have elapsed, when there may be a daily expectation of his recovery. The tongue becomes moist, pulse less frequent, diarrhœa ceases and no fresh spots appear; and the temperature again subsides in the same way as it rose.

If the patient do not recover, all the bad symptoms which I described to you in typhus become apparent, the patient lies prostrate on his back, delirious or muttering, tongue very dry, mouth covered with sordes, pulse 104 or more, retention of urine, abdomen much distended and tympanitic, the breathing is quicker, showing increasing congestion of the lungs, a general lividity of the extremities, but no petechial change of the rose spots. Death may occur from simple prostration, but there is generally some more special cause for dissolution; for instance, the diarrhœa may be excessive or the hæmorrhage may be large and so prove fatal, or a perforation of the intestine may take place. In other cases pneumonia is added to the congestion of the lungs, and in this way death may ensue.

When perforation occurs, it may be suspected, if the patient have not previously been ill by his becoming collapsed with distension of the abdomen. In bad cases, however, the diagnosis is by no means easy, for, owing to the existing extreme prostration of the patient, the lowered state of the pulse or increased tympanitic state of the belly may not be discernible, and on the other hand, from this increased distension and lowering of pulse being the concomitants of the typhoid condition, a perforation is sometimes suspected when it has not occurred.

On post-mortem examination of a fatal case of typhoid fever various morbid appearances may be found. There will be the characteristic changes in the ileum, as I have already described, the amount of deposit remaining in the glands varies with the time of death. At the usual period of death—between the third and fourth week—the Peyer's patches will be found having the rough and shaggy appearances before mentioned, with slough and fæcal matter attached. In other cases portions of the mucous membrane will have been destroyed, and veritable ulcers may be said to exist. If there has been fatal hæmorrhage the glandular patches will be seen to have undergone a more active sloughing, and perhaps an open blood-vessel may be found. If perforation has taken place, the mucous and muscular coats will be seen to have been involved in the sloughing process and the peritoneal coat perforated. Sometimes there is a good-sized rent, at other times a large surface of peritoneum is laid bare within, and perforation has occurred by a number of minute pinhole points, and thirdly the rent may have occurred from simple rupture. These rents are seen best on the external surface, and here also is generally found some lymph or as much inflammatory exudation as could be produced in the few hours of life after the rupture of the gut. Associated with the intestinal disease is a considerable enlargement of the mesenteric glands which are observed to be swollen and soft, so that they readily break down on pressure. The new material which forms in their interior is considered by some to be of a specific nature, resembling the typhoid matter in the intestinal glands. The spleen, as a rule, is two or three times its natural size. The lungs are congested at their posterior parts, and sometimes associated with the congestion there is an actual inflammation, so that the back part of the lung and that portion near the root are found consolidated from a combination of these two conditions. Occasionally another organ is affected—the larynx; at the posterior part of the

vocal cords, at the root of the arytenoid cartilages, a brown soft deposit is met with, which exactly resembles the typhoid deposit in the intestine. This sloughs out, and in some cases has led to perforation, or has been the origin of a chronic disease of the larynx.

The prognosis in typhoid is not as in typhus, according to the age of the patient, which, in other words, means in relation to his strength; for death occurs in typhoid from local or accidental causes, as hæmorrhage, perforation, &c., and the risks seem to have to do more with the virulence of the disease than the condition of the patient.

Perforation occurs in a quarter of all the deaths. In the remainder the cause is hæmorrhage, congestive pneumonia, or simple exhaustion.

Varieties.—The varieties of typhoid may be best remembered as of three kinds. There is the natural division into the *mild* and the *grave.* You may have one patient, for example, who has clearly all the symptoms of the disease upon him, and yet at the same time is never very ill, and passes though the complaint without a single urgent symptom; then, on the other hand, there is the patient who from the beginning is excessively ill, so that you watch the case throughout with the greatest anxiety. Then there is a third class of case which comes under neither category, where the patient has few symptoms or none at all, and yet the typhoid changes are going on in the intestine which may end in death. This *insidious and latent* form has been called by German writers *typhus ambulatorius.* (I might here warn you not to mistake the meaning of the word typhus when you read of it in foreign books. Both the French and Germans mean our "Typhoid or Enteric Fever" when they speak of typhus. They have little of our true typhus, which they call the English disease or exanthemic typhus; as, however, they often qualify the word by the epithet "Typhus Abdominalis" there can be no mistake.) In this latent form of typhoid the patient may be so slightly ill that the real nature of the disease has been never suspected, when a fatal hæmorrhage may come on or a sudden perforation of the intestine.

Relapse.—Another remarkable circumstance about typhoid fever is that a relapse may occur. After the patient has lost his fever, and been recovering during two or three days, he again falls back into his original condition, in which the diarrhœa recurs, and a fresh crop of spots appear on the body. This second attack is of

varied duration. If he should die it will be found that a second typhoid process has been taking place in the glands of the intestine.

Causes.—It is generally believed that typhoid is due to the taking into the system a specific germ or virus, and that a reproduction of this takes place in the intestinal glands from which part germs are thrown off. As intestinal discharges eventually make their way into the drains and sewers, so it is supposed that through these channels especially the disease is propagated. Either some article of food has been contaminated by water having an impure source or the patient has breathed into his lungs some floating germs which have arisen from some polluted water. There is no reason to believe that any vaporous emanations from the body or lungs of a patient suffering from typhoid contain the poisonous element, and therefore typhoid is not contagious or infectious in the ordinary sense of these words. In nearly all the epidemics of typhoid which have lately occurred a source of pollution has been found either in the water or milk which has been used by the patients, hence the prevalent belief that the germs of the disease are carried about. Some, however, think that the typhoid poison can be self-generated, and therefore Dr Murchison has invented the word "pythogenic" as a substitute for typhoid, meaning "born of putridity," but as this might imply that any decomposing matter will produce the disease, which is not yet proved, I do not use the term.

Diagnosis.—I have already told you that a continuous febrile condition implies as a rule either the existence of a local disease, or a special virus working in the system. Now, as typhoid fever may sometimes occur without a rose rash or other very characteristic symptoms, you can easily see how it is that when neither local inflammation nor ordinary exanthemic disease can be discovered, a diagnosis in favour of typhoid is apt to be made. Thus it is that nearly every disease accompanied by fever is at times called typhoid. It is mostly due to carelessness, and therefore it would be a waste of time to tell you here how pneumonia, arachnitis, or peritonitis differ from typhoid fever. When I come to speak of these diseases separately you will see in what their peculiarities consist. Neither is there any very good reason why I should insist upon your endeavouring to distinguish typhoid from typhus. You are not likely to confound them, for, as a matter of fact, typhoid is far more often mistaken for local inflammations than for

typhus, but since the diseases were once confounded, it has become the fashion to treat of them together, to lecture upon them together, and to write upon them together. There is no reason in this, and it merely remains as a monument of our former ignorance. Why you as new men in the profession should be taught relatively to the shortcomings of your forefathers I do not know. As, however, you are constantly asked at examinations how to distinguish between typhus and typhoid, I will tabulate the main differences for you :—

	Typhus.	Typhoid.
Incubation	Nine days.	Twelve to fourteen days.
Onset	Sudden.	Slow.
Duration	Two weeks.	Three weeks or more.
Premonitory Fever	Three days.	Nine days.
Eruption	Mulberry. Universal. Continuous. May become petechial. Appears on fourth day.	Rose. Scattered. In successive crops. Non-petechial. Appears on tenth day.
Bowels	Abdomen natural form. Stools dark.	Abdomen tumid. Stools ochry and characteristic.
Appearance	Torpor.	Countenance more bright. Flush on chest.
Tongue	More furred and brown.	Red, chapped, and glazed.
Contagion	Highly contagious.	Non-contagious.
Temperature	Rapid uniform rise.	Peculiar daily rise and fall.
Causes	In connection with poverty and diet.	In connection with defective drainage.

Treatment.—The experience of all medical men coincides with what theory would teach: that in "a fever" much support is required. If it be true that a patient with fever is undergoing as much waste in twenty-four hours as if the subject of it had been walking all day without food, you will see that a large amount of nourishment is required to compensate for the loss. Now, since the patient's stomach is not in a condition to digest solids, the substitute which contains all the elements of nutrition is milk, and this you will find is the best diet for most of your cases. My hospital patients have four pints of milk a day, and I find they are much better cared for than many private patients whose stomachs are filled with beef tea, arrowroot, jelly, brandy, wine, and a host of other messes. If the patient cannot digest milk, then substitute

beef tea for a part of it. There is such a thing as too much feeding done, especially in rich families, where, owing to the doctor having told the nurse that the case is one for food rather than physic, she thereupon "pours in" all the nourishment she can, overloads the stomach, which, unable to get rid of its contents, becomes paralysed, the ganglionic nerves receive a shock, and the patient is restless and prostrate until he has vomited all he has taken, when the pulse rising he becomes calm and sleeps. This is a practical hint worth remembering—that a patient with an overloaded stomach is far more depressed than he is with a long-continued empty one. You need not give your patients stimulants as a matter of course. That alcohol tends to cure fever by antagonising any virus in the system is a mistake; in a large number of cases the average number of cures is higher without stimulants than with them. They are, however, of eminent service, and we judge of their necessity by the debility of the patient, and can test their influence by looking for the subsidence of outward symptoms. A weak action of heart, very quick pulse, dry brown tongue, delirium and such symptoms suggest their use. We also use stimulating and tonic medicines. Ammonia is often good, and sometimes quinine acts admirably as being instrumental in lowering the animal heat. If the diarrhœa be excessive we usually give an opiate enema, but it must be remembered that looseness of the bowels is a symptom of the disease, and cannot be checked by the administration of medicine. If much restlessness a mild opiate, such as Dover's powder or opium enema. If there be much hæmorrhage from the bowel the ordinary remedies are useful, as gallic acid and lead. If you should be called to a case—as I have been—when an immense amount of blood has been passed and the patient is blanched and pulseless, the best thing is to inject per rectum half an ounce of turpentine and repeat it. It acts as a stimulant, and being absorbed into the system tends to check the hæmorrhage.

RELAPSING FEVER

Relapsing fever was not recognised in London until the time of the Irish famine thirty years ago, but since this period it has occurred here twice as an epidemic. It is essentially connected with famine and destitution, and has therefore been styled "famine

fever." It is not a very fatal disorder, and its peculiarity consists in the total abatement of the symptoms, and their recurrence in a few days.

It is contagious, and like other forms of disease of the same kind is supposed to be due to a specific virus. The accession of the febrile symptoms is sudden, often attended with vomiting and delirium; the temperature may rise to 106° in twenty-four hours, although it usually falls again two degrees. The patient is by this time exceedingly depressed, his eyes are sunken, and he speaks with a feeble voice, but the brain is clear. The tongue is furred or slightly brown. There is pain in all the limbs, and this is often complained of bitterly by the patient as his worst symptom; there is occasionally slight jaundice.

These symptoms last on an average a week, then quickly subside. The fall of temperature in some cases is so sudden that the patient is momentarily collapsed; thus I have had two cases of the kind in which the temperature fell in a few hours from fever heat to 2° below normal. After this subsidence of fever the patient is comparatively well, and often leaves his bed; but the period is of short duration, for on the fourteenth day he again suddenly has a recurrence of the febrile symptoms, the temperature rapidly rises in twenty-four hours, and the patient is in bed and as feeble and prostrate as ever. The time of relapse appears to be very definite; the period of the first paroxysm and the subsequent intermissions vary, but the two periods together almost invariably occupy fourteen days. Thus, if the stage of fever were five days, that of the intermission would be longer; if the stage of fever were eight days, then the intermission would be shorter. When, on the fourteenth day, the febrile symptoms recur, they continue only for about three days and then quickly subside. After this the patient gets well. He may, however, have a second relapse of shorter duration, and even a third relapse.

The remarkable nature of the complaint could never have received a better illustration than it did at this hospital three years ago. One of our pupils had been appointed resident medical officer at a fever hospital, and being very assiduous in his duties soon caught relapsing fever. Being very ill he desired to be brought here, when he soon got better, and in a few days was able to leave his bed. This was on a Saturday, when there happened to be a debate at the Physical Society on Relapsing Fever. Our patient went to the meeting, entered into the discussion, and to the

surprise of his audience, said that at that moment he was the subject of the disease, that although he then felt pretty well, he would be found in the Clinical Ward on the following Tuesday with intense febrile symptoms, and all the other indications of the disease. On the day mentioned he was found as predicted, lying in bed in an extreme state of prostration, and the most violent symptoms of the disease upon him.

The disease is rarely fatal, except in places where it is epidemic, and great impoverishment exists. There is no rash, but in some cases there appears a tendency to a purplish mottling on the abdomen.

There was no difficulty in the diagnosis at the fever hospitals, owing to the sudden accession of the symptoms and the absence of the mulberry rash. The first fact excluded typhoid, and the second excluded typhus.

SCARLATINA

This is the worst form of specific disease existing in our country. It is always more or less amongst us, and often puts on the most virulent character. The mortality has sometimes amounted to 30,000 annually, but its evils do not end here, for, owing to its various complications, it may be followed by kidney disease, arthritic affections, deafness, and perhaps abscess in the brain. Although it is the most fatal and formidable of diseases, yet it may assume the mildest form, and patients may go through its stages without being aware of the nature of the slight ailment which affects them. In schools and public establishments every variety of the disorder may be observed between the most virulent form on the one hand, and a disorder characterised only by a slight sore throat on the other. This has given rise in the public mind to an opinion that there is a mild variety—"scarlatina"—to be distinguished from the more severe "scarlet fever." I would ask you never to assent to these popular errors as medical men sometimes do, it being your duty to instruct your patients and not to assist in perpetuating their delusions.

In its simple form it runs a more rapid course than any other of the specific diseases. The incubation is from three to six days, but several cases have been recorded where it is less, as, for example, where a child has come from school with the disease upon him, and

in twenty-four hours afterwards some of his brothers and sisters have sickened with the complaint. The febrile symptoms sometimes are of a most violent kind, whilst in the milder form of the disease they are scarcely noticeable. In the former case they may be ushered in by delirium and convulsions, intensely hot skin, thickly-coated tongue, very quick pulse, and at the end of twenty-four hours—as it is seldom before this that the thermometer is used—the temperature may stand at 104° or 105°. At the expiration of this time the rash has appeared, in fact the whole of the surfaces of the body, both without and within, are in an intensely hyperæmic condition. And, now, I would warn you not to seek for the first appearance of the rash upon the face, for if you do, and the child be wrapped up in bed, you may entirely overlook its existence. You will generally first notice it about the neck, and then on the trunk, and soon you will see the whole body, including the limbs, and perhaps the face, covered. You will now find the body as red as a lobster, or as if the patient had been put in a hot bath and violently rubbed. Observed at some distance, the skin looks uniformly red, but on closer examination it will be observed that the colour does not seem as if it had been painted smoothly over, but as if scattered in minute points over the surface. The eruption, indeed, is made up of innumerable very minute points. Sometimes the whole surface is not affected, but the rash is seen in parts only, as on the chest or abdomen, and, at the same time, on portions of the legs. In less severe cases, some patches of red rash are seen, and often on the flexures of the knees and about the ankles. In simple cases of scarlet fever, the rash is a mere exanthem, and therefore fades on pressure, but there often accompanies it a papular rash, causing the skin to be quite rough when the hand is passed over it, and sometimes creating a suspicion of the onset of smallpox. Not infrequently, also, some of these papules undergo a further stage of inflammation, and distinct vesicles are formed. Last year I saw two cases where so large a crop of vesicles came out on the forearms that it suggested to the medical men a complication of smallpox. The scarlatina rash lasts four or five days and then fades, beginning to disappear at those spots where it first began. Then desquamation commences by an universal shedding of the epithelium, and sometimes as regards the hands by the throwing-off of the cuticle like a glove. The desquamation, I must tell you, is not peculiar to scarlatina, but in this disease it occurs in an excessive

degree. There is never an extreme congestion of the capillaries of the skin without a necessary death of the epithelium and a consequent shedding of it. In all erythemas naturally or artificially produced this may be observed.

Now, I told you there was every reason to believe that the internal, mucous surface was affected in a similar way to the external, and thus when the rash begins to appear, it may also be observed that the mucous membrane of the mouth is intensely red from capillary injection. The tip of the tongue is covered with minute red points, and the papillæ may be seen projecting through the white fur. The soft palate and tonsils are red, swollen, and covered with a tenacious secretion. The glands beneath the jaw are swollen, forming a large disfiguring mass in the neck. The congested or inflamed condition of throat often extends to the back of the nares and even along the passages of the nostrils, by which they become blocked with secretion, which is often of an offensive nature. The inflammation also involves the Eustachian tubes, causing great pain in swallowing. If the patient has been sick, some casts of gastric follicles have been found, showing how the whole alimentary tract is involved. The child, in a moderately severe case of scarlet fever, remains in a most deplorable condition for four or five days—the high temperature is kept up, skin dry, great swelling of neck, difficulty of swallowing or of breathing, on account of the swollen and inflamed state of the throat and nares, and from these perhaps there is a discharge. Desquamation then takes place, and recovery ensues. If the urine is carefully examined there may sometimes be found in it some tubular epithelium.

The case I have described may be called (1) *Scarlatina simplex.*

The form I have alluded to where the patient is unconscious of having had the disorder is called (2) *Scarlatina sine eruptione.*

It sometimes happens, and these cases are of the most fatal kind, that the rash is slight, but the whole virulence of the disease is concentrated on the throat; the neck externally becomes enormously swollen, suppuration may occur, and sometimes sloughing, whereby the whole of the skin of the neck is destroyed, and death then sometimes ensues from implication of the larger blood-vessels. This is the (3) *Scarlatina anginosa or faucium.*

And now I must draw your attention to the affection of the kidney, which is so often a complication of scarlatina. You know that dropsy very frequently follows scarlatina, and that the urine

becomes brown, of high specific gravity, and contains inflammatory casts, epithelium, and blood globules. It had long been observed that this dropsy came on from ten to fourteen days after the onset of the fever, and that the latter had been so slight that the patient had scarcely kept his bed; therefore, the opinion naturally arose that the nephritis was due to cold acting on a person who had not paid sufficient heed to his disorder. Such an opinion is now held by many, yet against this view must be placed the fact of the excessive proneness of scarlet fever subjects to dropsy; as it is sometimes seen accompanying the worst forms, and even occurring at the onset of the disease, whilst the patient is kept warm in bed. It is therefore thought, and this is my own opinion, that the kidneys are prone to epithelial changes, ending in inflammation, in all varieties of scarlatina; that in the scarlatina simplex, little more occurs than a slight desquamation of epithelium, whilst the force of the disease is expended on the skin; that in the scarlatina anginosa the whole virulence of the poison is concentrated on the throat, but that there is another form in which all the outward manifestations of the disease are slight, but the whole violence of the disease is thrown upon the kidney. In this case of course the patient would have been thought to have had the disease mildly, but the epithelial change is meanwhile going on in the kidney, until at last the tubules become choked, the circulation arrested, the character of the urine changed, and dropsy appears. The latter, therefore, would not be evident as a rule until some days had elapsed. If the patient had been out and exposed to cold this might be an additional exciting cause for the development of the nephritis, but I cannot look upon cold as the originator of it. You may remember that nephritis occasionally is associated with other febrile disorders. If this be true the renal disease should not be regarded so much a complication as a variety of the disease, and be styled (4) *Scarlatina renum.*

There is yet another variety of the disease (5), *Scarlatina maligna*, that form in which the poison is so intense that it kills its victim before the usual phenomena of the disease appear. He is no sooner taken ill than he becomes rapidly collapsed, pulse scarcely to be felt, the whole surface of the body livid, general torpor, and death in a few hours. Perhaps petechial spots may appear on the legs, or some hæmorrhage from the mucous membrane. If there is no history of contagion, the rapidity of the illness will scarcely leave a doubt as to its nature. For example, I lately saw a boy who was playing with his school-fellows on Saturday afternoon, complained of being

ill on Sunday, although he was up. On Monday morning the medical man was sent for, and found him collapsed; in the afternoon I saw him, he was then dying, almost insensible, skin livid, hands and feet purple, and pulse 160. I said the only disease it could possibly be was scarlatina, and this opinion was confirmed by other members of the family subsequently having the disease.

As regards other symptoms and complications, there is the renal nephritis already alluded to, but a commoner complication than this is a secondary fever into which the patient falls. You may observe a large number of your cases suffer in this way. There are only one or two days of convalescence when the child again becomes feverish, the temperature being three or four degrees above normal, the skin very dry, tongue furred, and pain in all the limbs. Very often the joints swell, and the patient is said to have a rheumatic attack. It is thought by some to partake rather of the nature of pyæmia, and then its source is looked for in the sloughing glands of the neck, but since these are not always present the explanation is not satisfactory. It is remarkable that further complications are then liable to ensue, as pericarditis and endocarditis. Thus it is not unusual to hear a patient trace his heart disorder to a scarlatina suffered many years before. The arthritic affections may be only another phase of the scarlatinal disorder, and it is remarkable that the dengue of tropical climates is a complaint in which arthritic pains and a red rash are combined. With this secondary fever there is often a constant discharge from the nose and the ears, the latter sometimes leading to permanent deafness. Sometimes when the rash has appeared this secondary fever continues, and thus the temperature remains permanently high for three or four weeks.

Scarlatina is eminently contagious, the poison being given off from the surface of the body and from the lungs. The blood is also said to contain the virus, and of late it has been pretty satisfactorily shown that the epithelial scales contain the poison, and that if these are swallowed they will set up the disease. The case of a desquamating boy milking cows, and those who partook of his milk catching the disease, may be mentioned in corroboration. The poison attaches itself to articles of furniture for many months, and may be conveyed by clothes, and probably by letter.

The treatment is, in the first place, to procure as much good air as possible to surround the patient, and to give him cooling drinks. Stimulants according to circumstances.

MEASLES

Measles has an incubation of twelve to fourteen days, when taken in the ordinary manner. If the secretion be inoculated, the first symptoms of the disease appear in a week. The disease approaches with all the usual febrile symptoms, to which are added those of a severe cold or bronchitis. The face is puffy, the nose runs, the eyes are watery, and there is sneezing. The temperature rises to 104° or 105° in from thirty-six to forty-eight hours. After these febrile and catarrhal symptoms have lasted for three days the rash appears. This is first observed about the roots of the hairs at the forehead, and spreads from here to all parts of the body, being full out in another day. The temperature, then, is often observed to fall, standing at about two degrees above normal heat during the remainder of the complaint. This loss of fever is a fact well known to mothers and nurses, who are aware that children will often be able to amuse themselves as in health whilst the rash is out. The latter continues for four or five days, and then departs. It differs from that of scarlatina in not being so uniform, but in patches, these being disposed to assume a crescentic form. The colour is not so bright red as that of scarlatina, but is more yellowish-brown. It is often even darker, and leaves a brown stain for some considerable time after the rash has gone. During the height of the exanthem there is often associated with it a slight papular eruption as in scarlatina, but not to the same extent. When the rash is over, a slight desquamation takes place, but this is less than what occurs in scarlatina. The temperature has now fallen to the normal standard. During the progress of the complaint the swelling of the face and the catarrh continue. If the case be a mild one the catarrhal symptoms depart at the same time with the rash, and the child may be considered almost well.

In bad cases an actual bronchitis occurs, so that the danger of the disease lies in the pulmonary complication; therefore it is that measles is so rarely fatal amongst the better classes who are taken care of, whilst it is attended by so many dangers amongst the poor. The chest complication, which in the first place is a severe bronchitis, subsequently may develope into a catarrhal or broncho-pneumonia. This is what we find on post-mortem examination, and it is not because broncho or lobular pneumonia is especially a disease of children that this form of inflammation occurs, for the

same character of disease is found in adults, as we had an opportunity of observing here a few years ago in the case of some sailors; in those who died a true broncho or lobular pneumonia was found. In this bronchitis of measles very large quantities of purulent matter are expectorated, and if not immediately fatal, it may continue as a chronic affection, and the child at the same time wasting he may be thought to be in a consumption; recovery, however, eventually takes place. In a similar manner a wasting diarrhœa may continue for a lengthened period. Very exceptionally there may follow a nephritis and albuminuria.

Measles is highly contagious by means of the vaporous emanations from the lungs and body. The secretions from the nose are also highly contagious. The cause is generally believed to be a specific animal cell, although it has been suggested by Hallier to be a vegetable germ; in fact, an already recognised fungus.

There are no important varieties. A malignant form is spoken of as occurring in some parts of Europe, but I have never seen it. A variety is sometimes spoken of as black measles, where the rash is remarkably dark. I have on several occasions seen the rash as dark as mahogany, but the disease otherwise was not more severe.

The treatment consists in pure warm air, and cooling drinks.

HYBRID MEASLES

I ought to inform you that some medical men have regarded scarlatina and measles as varieties of the same disease, since sometimes cases may be met with where the symptoms partake of the nature of the two affections. Other observers, when meeting with cases of this kind, have considered that they have before them a combination of the two diseases. Others have regarded them as a variety of measles, and others again as a variety of scarlatina. The cases are those where sore-throat and swelling of the neck are associated with coryza and a rash, having an intermediate character between the two exanthems. A child, for instance, is observed to have the puffy face and sneezing of measles, whilst at the same time there exist great injection and redness of the throat, together with a swelling of the glands of the neck. On the third day a rash is apparent on the face, and becomes diffused over the body; it is patchy and red, and after four days is followed by desquamation;

dropsy sometimes occurs. During the disease the throat may be very bad, and there may be pulmonary complications.

There exists, therefore, a disease which in its symptoms partakes of the nature both of scarlatina and measles, having the throat of the former with the coryza of the latter, and a rash which is intermediate in character. I before told you that the test of the specificity of a disease is to be found in its mode of propagation, and therefore we must further ask whether this hybrid disease is found in connectiou either with pure measles or scarlatina? I believe it is not. I had an opportunity some years ago of seeing examples of this affection, and I noticed that several of the kind occurred in the same house. I am therefore led to believe that we are dealing with a distinct form of exanthem. Those who hold this opinion would style it "rubeola" or "rugeole," and so distinguish it from scarlet fever and measles, which would then be technically called "scarlatina" and "morbilli."

SMALLPOX

Smallpox is a specific contagious disease, commencing with febrile disturbance, and more especially with sickness, headache and pain in the back, followed on the third day by an eruption of pimples, which inflame and suppurate, accompanied by some similar eruption on the mucous membrane of the mouth.

Incubation.—The average time is twelve days, never longer than a fortnight, and never been observed less than ten days. When the variolous matter is introduced by inoculation, the system is affected in eight days; during this time the pustule is forming, and when it is quite mature constitutional symptoms ensue, and the general eruption makes its appearance.

The *initiatory fever* is often marked by the symptoms above named, and therefore the nature of the disease is at once suggested; I refer more especially to the sickness and pain in the back. The latter often is so great that it constitutes the patient's principal trouble, and has been the cause of his seeking medical advice. In children there is often delirium and convulsions.

Premonitory rash.—Not uncommonly a general roseolous rash precedes the true variolous eruption; this is a true exanthem, and may mislead you as to the nature of the approaching disorder. In some malignant forms of the disease, as well as in mild ones

where persons have been protected by vaccination, this rose rash may be the only cutaneous evidence of the existence of smallpox.

The roseolous rash, if it has been present, soon subsides, and makes way for the true eruption. This commences as a number of papules scattered over the body; they are seen first on the face, or perhaps about the same time on the wrists or backs of the hands, and then spread. Before the eye can perceive them they may be felt by the hand as if grains of sand were scattered over the surface. These papules enlarge, then some fluid is seen within them, constituting them vesicles; afterwards this becomes opaque, and they are styled pustules. It takes eight days for the pustule to form, and this is called the period of "maturation." These pustules are about the size of peas, and may cover the body in more or less degrees. When scattered and separate they yet have a tendency to form in groups of four or five together. The pustule is round, and depressed or umbilicated in the centre, which is characteristic of the disease. Most vesicles or pustules, as you know, are acuminated, but the variolous pustule is depressed in the centre. This seems to be due to the inflammatory products being formed around a hair follicle, with its sebaceous glands, and in the case of the palm of the hand at those points in the skin which would correspond to the site of these structures; consequently, instead of the secretion, which is formed in the rete mucosum, simply raising this up together with the cuticle in a pyramidal shape, it is confined in a circular space around a point where the cuticle and new membrane still remain attached to the cutis beneath. This string of attachment is called the stigma. The formation of the variolous pustule is not much unlike that of the vaccine-vesicle, and therefore you may get an idea of its structure from a knowledge of the latter. You know that in obtaining matter from this, you have to prick the vesicle all around, as the fluid is contained in separate chambers, and you know also how this is shown in the cicatrix which is left; a vaccine scar being circular, somewhat depressed, radiated and foveated, that is, marked with minute pits. Now, the smallpox pustule has much the same formation, it is not really divided into separate chambers, although the mode in which it is developed has a tendency to divide the secretion into portions. Thus, there is produced from the rete mucosum a softish layer of lymph, and this being raised at the sides whilst it is held down in the middle, a number of almost distinct spaces are formed by the folds and corresponding chambers between them. This umbilicated pustule is mature in eight days; it

then changes its colour, turns yellow with a faint redness around the margin ; it then becomes brown, with a distinct red halo around it, the filamentous attachment or stigma gives way, and the pustule discharges itself, the matter from which, together with the broken pustule, form scabs on the surface. These eventually fall off, leaving reddish stains or pits.

I have said that at the onset of variola the febrile symptoms run high, and thus on the third day the temperature may be observed as high as 105°, but afterwards it falls, and for two or three days before maturation is complete, the temperature has fallen to normal, at which point it is found on the eighth, ninth, and tenth day. Now, after this there is a second accession of fever, and herein you will observe a difference between variola and other exanthemata. It resembles them in the subsidence of fever with the departure of the rash, but in them the eruption is no more than an exanthem, whilst in smallpox there is an actual inflammation of the skin, which in itself is productive of important symptoms. Thus, as soon as the pustules are matured, and inflammation occurs around them, they burst, and the process is accompanied by the secondary fever, and a rise of temperature to 104° about the fourteenth day. This remains for two or three days, when it again declines and the patient is convalescent.

During the progress of the disease pustules may be observed on the tongue and inside of the mouth. The whole of the mucous membrane is much injected. At the same time there is a similar condition of the bronchial passages, with secretion from them. The eyelids are swollen, and the conjunctivæ inflamed. There is often delirium at night, and other symptoms already described as accompanying the fever of the exanthemata. The danger depends upon the virulence of the poison and the amount of the rash, also upon the degree of pulmonary obstruction.

You may remember that we divide a case of smallpox into four stages—that of incubation lasting twelve days, the eruptive fever lasting two days, that of maturation eight days, and the secondary fever three or four days.

Dr Gregory, the physician to the Smallpox Hospital, was in the habit of dividing smallpox into the following varieties :—1. Variola discreta ; 2. V. confluens ; 3. V. semiconfluens ; 4. V. corymbosa ; 5. V. benigna ; 6. V. maligna ; 7. V. anomala.

The *first* form is that where the pustules are distinct, so that the whole number might possibly be counted.

The *confluent* form is that where the pustules on the face and some other parts of the body are liable to run together; and this, excepting the malignant form, is the most fatal variety, as half the patients attacked by it die. As regards the face the pustules coalesce, so that it presents one whitish shapeless mass, like a bladder of lard, and it is only by careful attention that the outlines of the different pustules can be made out. Death often occurs before pustulation is complete, and therefore the central depression is often not well-marked. The disease is rarely confluent over the whole body, the coalescence occurring on the face and parts of the limbs. There is generally sore-throat, and much swelling of the neck externally. The inside of the mouth and tongue is also affected, causing much difficulty in swallowing, and the larynx and bronchial tubes are covered with secretion. In all cases of smallpox there is usually a very peculiar and disagreeable odour given off, but in the confluent form the fœtor is horrible. In these cases the cellular membrane may become affected as well as the true skin. The blindness which so often follows smallpox is not from the formation of pustules on the eye, but from conjunctivitis going on to inflammation of the deeper structures, and this may also occur during convalescence.

The *third* form carries its meaning with it.

The *fourth* form I have never seen, but it was said to constitute a very fatal variety at a former period; it was the case where the pustules were arranged in distinct clusters on the body.

The *fifth* form was the mild variety occurring after vaccination; and the *seventh* was smallpox in combination with other diseases.

The *sixth* form, the *malignant*, or black smallpox, is the most important and fatal of all the varieties of smallpox; it occurred extensively during the late epidemic in England, and I unfortunately saw many cases of it. I have already said that in nearly all these specific diseases, the poison, owing either to its peculiar virulence or concentration, may act so suddenly and violently on the system that it seems to destroy at once the vitality of the blood. This becoming quickly corrupt, bursts forth from all parts of the body, and death soon ensues. This is eminently the case in smallpox. There may be the usual premonitory fever, and then perhaps a roseolous rash appears. This, especially on the face, hands, and feet, becomes of a dark purple colour, the pulse fades away, death ensues, and the only evidence of smallpox is the history, as far as it has reached, and the concomitant circumstances. In other cases some papules appear, and here and there a vesicle which is beginning to show its

umbilicated form; these papules and vesicles become of a black colour; and large purple patches extend over the skin. The face is swollen and purple, the eyes bloodshot, and blood oozes from the mouth. Blood may also pass from the bowels and kidneys. It is a remarkable circumstance, which is so different form what is observed in typhus and some other disorders, that the mind of the patient is perfectly clear. It is always fatal, and long before the pustulation is complete.

On post-mortem examination in these cases nothing more is found than would happen with a dissolution of the blood. In ordinary and discrete smallpox pustules are found in the mouth and pharynx, but I am not aware that they exist down the alimentary canal. There may be found bronchitis, pneumonia, or pleurisy, and it is through the chest that death usually occurs in the simpler forms of the disease.

Contagion.—The disease is highly contagious, the vaporous emanations from the lungs and skin carrying the virus. The pustular secretions contain it as well as the dried scabs. It is also infectious before the eruption appears, during the time of the initiatory fever. In the late epidemic a patient with smallpox was admitted unwittingly into one of the London Hospitals, and the bed clothes becoming mixed with other linen the disease spread rapidly through the institution.

The matter from a pustule is seen to contain a number of particles which are the true virulent agents. In the case of the analogous sheep-pox Dr Klein has found fructifying organisms, as he has done in typhoid fever.

Treatment.—A large, airy room, cooling drinks, and good nourishment. In the worst forms stimulants, as brandy and egg. If the patient is restless and sleepless an opiate is very useful. Various remedies have been suggested, as modifying or averting the disease, such as sarracenia, strychnia, &c., but hitherto have been used without success. Since it is evident that the danger is to a certain extent proportional to the amount of pustulation, and since the latter is greater on the face, hands, and exposed parts of the body, it has been thought that the development of the pustules is favoured by the influence of light, and therefore that this would be retarded were the patient placed in the dark. The practice has been followed, and it is said with success; in patients shut up in an absolutely dark room, the extreme pustulation has been prevented, and the accompanying symptoms reduced in severity.

It has also been thought that if the pustulation can be retarded by local means it would be beneficial, and thus in the Paris hospitals the nurses often prick the pustules, and insert within each of them a point of nitrate of silver. Then, also, at different times various applications, as mercurial ointment, have been used, and of late years a layer of gutta percha dissolved in chloroform. I have adopted this plan with no marked success. We are usually content to apply a little oil, in order to counteract the unpleasantness of the scabbing and drying process.

INTERMITTENT FEVER OR AGUE

Marsh Fever or Malarial Fever.—The malaria is a product of vegetable decomposition, requiring moisture and a certain temperature. The great peculiarity of the fever produced by this malaria is its intermittent character. In our own climate the fever is not a dangerous one, but in the tropics, where the rains are great, the temperature high, and vegetation luxurious, the fever is often of the most deadly character. The intermission even may be wanting, so that the fevers are often styled remittent, and in some cases almost resemble the continued fevers. Thus, most of the Indian fevers, as the jungle fever, are malarious, and their nature is expressed by the term "fever and ague," as the disease is designated by most of our seafaring men. It is probable that the fevers of the coast of Africa, of the Mauritius, and even the yellow fever, are of malarious origin, but I will say nothing further upon them, seeing that they have never been studied with the precision of the fevers which prevail in Europe. As regards England a large part of the eastern coast was at one time aguish, but thanks to better drainage the disease is much less rife than formerly. The bank of our own river Thames was also a principal source of malaria, and even now, below London, ague may frequently be met with; in fact, the whole of London, at one time, was excessively aguish, as you may learn from the writings of Sydenham and others; and, if I remember rightly, more than one royal personage is said to have died in consequence. William the Third is reported to have succumbed to ague, and when you read of the success of bark in fever it was, no doubt, in counteracting ague rather than typhus to which it owed its fame. At the present time if the earth be overturned a malarious poison is exhaled; and the proof that this lay in the soil was found in the very remark-

able circumstance that during the formation of the suburban railways ague was rife amongst the excavators or "navvies," and even in so elevated a spot as Sydenham several cases occurred amongst the men who were laying the foundation of the Crystal Palace. Under ordinary circumstances and when the poisonous exhalations spontaneously rise, they remain confined to the hollows, and do not rise to the surrounding hills.

As you might suppose, the disease is more rife in the autumn when decomposition of vegetable matter takes place, but it may lie dormant all the winter and break out again in the spring. It is mostly in the autumn that we see ague at Guy's Hospital, for at that time a large number of men and women proceed to the marshy districts of Kent for the purpose of pea-picking and hopping, and lying out under the hedges all night are very apt to acquire the disease. It is at this time that so many come to the hospital; and now note what is very remarkable in this disorder, that the poison may remain latent for a very considerable time, and then break out again in all its virulence. Thus, these poor people of whom I speak, after returning to London, apparently rid of the disease, may be living here in Bermondsey or elsewhere during the winter and seem to be well, yet, as soon as the warm weather in the spring comes on, the old enemy makes its appearance. You may have constantly seen persons in our out-patients' room present themselves with ague in the early spring, saying they had been ill a week or two only, and had been resident in London all the winter, but that in the preceding autumn they were exposed to the malarious influences before named.

You will constantly observe, too, that persons who have suffered from intermittent fever in India have fresh attacks during their sojourn in England, the poison having been lying latent in them for many months.

The malarious poison appears to have various degrees of virulence; you know that a person sleeping in the Pontine Marshes on his way to Rome, has often had the disease fully developed on the morrow; whilst in other places a long residence is necessary in order to receive into the system an amount of poison which may be injurious. A particular spot, too, may be more pernicious at one time than at another. For instance, in the Peninsular war, our men were often encamped on the side of a river and enjoyed good health, but after a hot season the rivers became dried up, and then the men who passed over their beds got severe attacks of ague; showing too

that the malarious influence is contained in the air, and is not produced by vicissitudes of heat and cold, as some have maintained; the poison is evidently carried by the wind, so that persons travelling on one side of a marsh may escape, whilst those on the other side may be attacked; and it has happened that a ship's crew have been affected when the vessel has been placed to windward of an infected coast.

It is very remarkable that animals seem to escape the influence; at least, they pasture in the most pestilential marshes with impunity, from which it appears that man alone is susceptible to its effects.

The most marked effects of the malaria is seen in the production of the ordinary paroxysm. This many of you probably are quite familiar with.

The ordinary paroxysm is divided into three stages: the cold, the hot, and the sweating, with a period of repose.

Now, we will suppose that the patient has a tertian ague, or that form where the attack occurs every other day, and that on one day he has been well. On the following morning the paroxysm appears. This *cold stage* may come on suddenly, but generally it approaches slowly by the patient feeling very ill, with lassitude, sickness, &c., followed by a sense of coldness in his back, which creeps up his spine until he feels chilled all over, and his whole body begins to shake, his teeth chatter, and his very bed creaks with the motion. The skin is dry, and you see the hair bulbs elevated, producing the "cutis anserina," or goose's skin. The face is pale or slightly livid, and the man looks cold. The pulse is feeble, showing that the circulation is interfered with. There is sometimes sickness. The urine is abundant and watery, and sometimes a constant desire to pass it. The patient feels so cold that he often asks, even in a tropical climate, for more clothes to be piled upon him; and yet, most remarkably, the patient is not actually colder, the sensation being altogether subjective. Not only is he not colder, but he is actually hotter; for the thermometer placed in the armpit may reach 104° or 105°. This cold stage may last an hour or two, or, we might say, from half an hour to two hours, and is succeeded by the *hot stage*. The patient now feels painfully oppressed by the heat, and the pulse rises and is fuller. The temperature now really is sometimes raised, being two or three degrees more than it was before; though this is not always the case. The skin is hot; the eyes bright; there is often headache and sometimes delirium; want of appetite as before; the urine more coloured; this hot stage generally lasts about two or three hours, and is followed by the *sweat-*

ing stage. A perspiration begins on the face, and extends over the whole body with great relief to the patient. The headache and other severe symptoms of oppression pass off, the pulse goes down, and the urine, which is now passed, contains an abundance of lithates. The patient falls into a sleep, and the paroxysm for that day is over.

Between the paroxysm there is a period of repose, but if the disease be severe the patient may feel ill in this interval. You may observe that since the thermometer has come into use, it has been found that the temperature begins to rise with the commencement of the fit, and continues rising until the completion of the hot stage, so that during the period in which the patient feels cold the thermometer shows a rise of three or four degrees. You may notice also that the paroxysm occurs mostly in the morning, and in this respect is distinguished from hectic fever, which occurs towards night.

The natural tendency is for the paroxysm, that is, the cold stage, to appear earlier every morning. If, however, medicines are given to retard it, then the paroxysm may appear later.

I told you that we had endeavoured to show that a relation exists between the febrile condition and the amount of tissue change, as represented by the solid ingredients of the urine. After the paroxysm in ague the urine is in large quantity and highly acid, supposed to indicate a critical change. It is said that uric acid, urea, and the chlorides are increased in quantity.

The varieties of ague depend upon the periods in which the paroxysms occur; if these happen daily the case is styled *quotidian*, if every other day *tertian*, and if every third day *quartan*. If a paroxysm should happen every day, but the times of occurrence correspond only on alternate days, the patient is said to have a *double tertian*. The variation depends probably upon the concentration of the poison, and this may be the reason why quotidian ague is more common in tropical climates than with us. In England the tertian form is by far the most common; indeed, we scarcely ever meet with any other variety. Quartan is rare, I believe, in any country.

The tendency of the disease is to a spontaneous cessation when the patient is removed from the marshy influence. The fact I have proved more than once in this hospital. Whilst the patient is undergoing cure the disease becomes less severe, the stages are less marked, the cold stage gives place to mere chilliness, and the sweating to a slight outbreak of moisture on the skin.

I should have mentioned that in ague the spleen becomes enlarged. This is not an hypertrophy but a mere engorgement, for as the patient is cured the organ regains its natural size; indeed, it becomes rapidly smaller under the influence of quinine. In tropical climates the spleen is so much enlarged and soft in persons subject to miasm that it has often been known to rupture by a very slight blow. Of course so remarkable an affection has received the attention of many theorists, and as long ago as Galen the paroxysms were considered due to some alteration in the spleen. Some have thought that a poison is received into this organ, and that a certain portion passing daily into the blood there undergoes some decomposition, giving rise to the paroxysm, by which way it is eliminated from the system. In connection with this theory it may be remembered that violent rigors are produced in pyæmia when morbid matters enter the blood, and that the same are often witnessed at the onset of the exanthemata. In cholera, too, when injections have been used for throwing fluids into the blood most violent shivering fits have been produced.

Then, besides the production of the special paroxysm there is the effect of the miasm on the general system of those who are always under its influence. They become chloro-anæmic, and are said to have a deficiency of red globules in the blood; their colour, however, is not so much pale as yellow or sallow. Both spleen and liver may become permanently deranged, and even, subsequently, dropsy may ensue. The most remarkable circumstance in connection with this poisoned state of the system is that the spleen appears to break up the red globules, and the hæmatine is carried into the tissue, producing a blackening of all the organ. This condition is styled melanæmia, and a good example of a brain so discoloured may be seen in 'Bright's Medical Reports.'

Diagnosis.—This is most important. Since, in the formation of abscess, in pyæmia, and in urinary disorders, paroxysms of shivering are very common, you may easily deceive yourself into the belief that you have a case of ague before you. A mistake of this kind is by no means uncommon.

Treatment.—A very favourite method has been to give a large dose of quinine before an expected paroxysm and so arrest it, and afterwards smaller doses to complete the cure. Of late years the hypodermic injection of medicines has been found very effectual, as in many instances the action of the remedy is more powerful and speedy than when given by the mouth. Thus, in the treatment of ague a

smaller dose of quinine is required, and this is important in countries where the drug is scarce and expensive. The plan is to dissolve six grains in a drachm of water with a drop of nitric acid, and then inject a quarter of this beneath the skin.

Arsenic also is a remarkable remedy, and is more especially useful in old ague cases which have long been subjected to the malarious poison. In wasted cachectic persons who have suffered from the long-continued agency of ague, the good effect of arsenic is sometimes most extraordinary.

DIPHTHERIA

Diphtheria is one of the most formidable maladies which we have of late years had to treat. It has probably always existed in a minor degree in all parts of the world, and occasionally spread as an epidemic under the name of the Egyptian malady or morbus strangulatorius. In our own country Dr Fothergill had treated of "cynanche suffocans," and since his time we have constantly met with cases, to which we could give no special name, but, being regarded by the light of the present day, were unquestionably cases of diphtheria. Bretonneau first gave this name, but it still remained unrecognised in England, until 1855, when the disease was imported from Boulogne. So little was it then known that the physicians on this side of the Channel regarded the complaint prevailing on the opposite shore as a malignant variety of scarlatina, but when the first case was imported to Folkestone its true and distinct character was seen. From this focus it spread first along the eastern counties, and then through the country generally, apparently quite irrespective of soil, impure atmosphere, or drainage. As regards London, it has been more frequently met with in the better class of houses in the suburbs than amongst the lower and dirtier habitations of the poor.

It is called diphtheria because a skin-like membrane forms on the throat and parts affected. The child (for it is a disease more prone to attack children) is supposed to have taken cold and complains of a sore throat; on looking at this part it is seen to be red and swollen, and at the same time there may be some slight swelling of the lymphatic glands beneath the jaw. In a very short time you perceive some white specks on the soft palate and along the edge of the uvula, or between this and the tonsils, or perhaps

one tonsil is much more swollen than the other, and on it is seen a white patch of secretion. In a few hours this has increased, covers a larger space and is seen to penetrate the mucous follicles; shortly it forms a distinct membrane which may then cover the tonsils, soft palate, and extend along in front on the roof of the mouth. The edge of the membrane is often free, so that if you tear it off you may have a cast of the whole back of the throat; the mucous membrane below being left red, vascular and bleeding, but not ulcerating. The membrane itself is tough and resembles wet wash leather; on its under surface you find epithelium stripped off from the mucous membrane, and the membrane itself is composed of fibrillated lymph mixed with inflammatory corpuscles. After the first appearance of the white secretion you may remove such a membrane in from thirty-six to forty-eight hours. In adults it takes longer to form. If it begins on one tonsil it spreads from it as a centre, and may put on a circular shape. The neck is generally much swollen from the enlargement of the glands beneath the jaw, but scarcely enlarged to the extent seen in scarlatina. At the onset of the complaint there may be febrile disturbance, but this soon passes off, and the skin becomes cool, and the pulse feeble.

If the membrane has been removed it will again form, and if not removed it becomes still thicker until it consists of distinct layers. As the new layers are forming from the mucous membrane the superficial ones are softening and decomposing, and to this particles of food adhere, so that if you now look into the throat you see shreds of rotten tissue hanging down and giving forth a most offensive or fetid odour. The term gangrene is a misnomer, as the parts do not really mortify. This horrible odour will at once suggest the nature of the case as soon as you enter the sick room. The membrane may go on forming for some time, and then if the process ceases recovery may occur, or it may spread still further along the posterior nares to the nostrils, or downwards to the larynx and air passages, when the additional symptoms of croup appear, and occasionally also the membrane extends down the œsophagus. The extension to the larynx constitutes the most important form of the disease which we meet with; this is first indicated by the child getting hoarse, and then having a harsh dry ringing cough, followed by evident obstruction in the breathing. The respiration becomes quicker, more difficult, the chest does not expand fully, and a lividity may be seen coming over the surface of the body. The child may have paroxysms of dyspnœa, as in

ordinary croup; fights for his breath, clutches at objects around for fear of suffocation, and at the same time has a loud crowing cough. Portions of membrane may be expelled, and our hopes are then raised for its complete removal.

In other and fatal forms, especially where the disease is epidemic, the diphtheritic process extends up the nostrils, and the most fetid discharge takes place from the nose. The neck is much swollen, and sometimes red, as if erysipelas were about to appear. The pulse generally is very feeble, and the extremities are often cold, and under these circumstances the child dies away from mere exhaustion.

You see, then, there are all degrees of the complaint, commencing with so slight ailments, that the nature of the case would not be known had not a small patch of membrane been accidentally discovered on the throat; and yet it is very remarkable as showing the constitutional nature of the complaint, that in some cases, where the local affection of the throat has been but trifling, the bodily depression has been most alarming: or if it is not primarily constitutional, it shows how soon the system can be infected from a local source. It is not, therefore, uncommon to see patients die in diphtheria from the constitutional depression.

Sir W. Jenner in his lectures made a division somewhat as follows:—1. The mild or simple form where the local affection is slight, and the fever small. 2. The throat variety where the disease commences in the throat, and then spreads. 3. The form where the first symptoms are those of croup. 4. The nasal form. 5. The Asthenic form, where the constitutional symptoms are out of all proportion to the local. A simpler division is that which every one naturally uses in describing his case, viz., the mild, malignant, and croupy. This diphtheritic complaint had not long prevailed in England, when it was observed by Dr Wade, of Birmingham, that the urine was frequently albuminous, and contained granular and epithelial casts; but it is remarkable that dropsy rarely follows.

The diphtheritic membrane need not be confined to the parts mentioned, for it sometimes affects the eye or an abraded surface on the skin, and I once saw a dreadful case where the diphtheritic process affected the pudendum in a woman after her confinement.

If you examine the mucous membrane after death it is found covered with shreds of exudation, the surface fissured, soft, and swollen. Sometimes, when cut through, a little purulent matter may be found beneath, but the mucous membrane is not ulcerated.

The membrane may be found covering the back of the tongue and all the parts before mentioned, and extending down the air passsges as far as the bifurcation of the trachea, where the membrane gradually melts into a muco-purulent secretion; although sometimes it may extend into the smaller bronchi, and the lungs themselves may be the subjects of lobular pneumonia. The kidneys are also liable to acute inflammation. It has been said that the blood has been found dark and thin, but this requires confirmation.

Diphtheria generally attacks healthy children, but it is found sometimes in connection with scarlatina, pneumonia, enteric fever, and other complaints.

It is a contagious disease; several instances have been recorded of medical men catching the complaint from infected children having coughed into their faces. This fact has given rise to the opinion held by Trousseau, Jenner, and others, that diphtheria is not so much a specific blood disease as due to a local specific poison which afterwards contaminates the system. It is not that the patient imbibes a poison like that which emanates from persons who are suffering from the exanthemic fevers, and that the local affections which accompany them are manifestations of this, but rather that the virus is implanted on the surface, thence spreads, and contaminates the system. A theory of this kind suggests the importance of destroying the first trace of the disease directly it makes its appearance.

I have told you that the larynx and trachea may be the only parts affected, and in this case the disease in appearance and symptoms resembles croup, or that membranous inflammation of the larger air passages which has long been known by this name. It is therefore asserted by very good authorities that the long and well-known croup which had always existed in this country was nothing more than diphtheria in a less virulent form. Croup and malignant throats were terms sufficient to designate the complaint until it became more virulent and epidemic when a special name was invented, and it was believed that we had a new disease. Many, however, will not admit this view, but maintain that croup is not diphtheria, that a membranous formation may arise in the larynx and trachea from cold without any specific cause, and that it may even occur from purely local irritation, as a scald; also that it is not attended by the constitutional disturbance witnessed in true diphtheria, viz., the albuminuria and remarkable nervous depression. The question is still a vexed one.

This depression of the nervous system which so often occurs in diphtheria is certainly most remarkable, and constitutes one of the most striking features of the complaint. It may accompany every variety of the disease, and be the actual cause of death by asthenia. A patient, for example, may be suffering from the ordinary form of diphtheria when he may become suddenly prostrate or collapsed. A late house surgeon who was apparently doing well with a doubtfully diphtheritic sore throat suddenly felt faint, and his pulse fell to 32. He never rallied, and died in a few hours. I have seen a child sufficiently convalescent to be up and dressed, and afterwards die in this collapsed state. In nearly all cases of diphtheria this tendency to depression may be observed by the low pulse and cool skin. In other cases it may show itself mainly in a weakness of the muscles of the throat when swallowing becomes an impossibility, the food being rejected through the nose. The most striking examples of this nervous affection are seen in persons after the acute diphtheritic process is over, when the whole body may be paralysed. Not only have they difficulty in swallowing, but the voice is altered, and the limbs are so weak that the patient may be unable to stand or to feed himself. The paralysis may affect also the muscles of the eye, the bladder, and other parts. It is, indeed, universal. Recovery slowly takes place. I remember the case of a young man who for many months after an attack of diphtheria had difficulty of swallowing and speaking aloud, when one day after exposure to cold all these symptoms were aggravated until he very rapidly had complete paralysis of the pharynx and larynx; he could not swallow, and his voice was stridulous from closure of the glottis. He very shortly died suffocated, and the post-mortem revealed no morbid change.

Treatment.—The first thing to consider is the propriety of local treatment, and as there are good reasons to believe that the disease may be implanted in one spot, and from thence be propagated, we should at once try to destroy the virus at its source. Nitrate of silver is always at hand and is very often used, but liquid escharotics are more penetrating and useful. From my own experience I believe there is nothing better than nitric acid applied with a glass pencil. If the surface is extensive and the acid cannot be well circumscribed, we use less violent remedies, as permanganate of potash, carbolic acid, and Tr. Ferri. I do not know that the first has the same power in preventing the spread of the virus as the second, which is therefore to be preferred; and as regards the iron, I know

that adults who have been able to express an opinion have felt more relief from this than any other application. You may, therefore, apply the strong tincture to the part, and let the patient wash his mouth with a diluted solution. As regards internal remedies, the profession is well agreed as to the need of support in the shape of milk, beef tea, and wine or brandy. For medicines the iron is probably the best, so that the child may use it for a gargle, and at the same time as a medicine. There are strong advocates for chlorate of potash and chlorine water, and if these are used you mix together chlorate of potash and hydrochloric acid. If unfortunately the trachea is involved a fatal result almost inevitably ensues, the child is too ill for an emetic, and at the same time the extension of the process may be deep into the bronchial tubes, and probably the patient is the subject of nephritis. It is not surprising, therefore, that the operation of tracheotomy is so seldom successful. In all probability the successful cases of tracheotomy for croup are cases of simple catarrhal laryngitis.

DISEASES OF THE RESPIRATORY ORGANS

PHYSICAL EXAMINATION OF THE CHEST

Before taking the diseases of the chest individually, I shall occupy your time for two or three lectures on the subject of physical examination. This method not only includes auscultation and percussion, but the shape of the chest and its movements. Indeed, there is no fact in connection with its formation and action which is not important, and therefore writers have done well to discuss them in all their particulars. As, however, I do not wish to burden your memories with any but the most important facts, I shall say nothing but what you may easily remember.

I will first write you out the following list of the leading physica signs on which we rely; these include—inspection, palpation, men suration, percussion, auscultation.

1. Inspection
   - Form of chest
     - Small; pigeon, narrow, flat.
     - Large; barrel shape.
     - Inequality of sides.
   - Mode of expansion.
     - Respiration imperfect on one side or part;
     - costal, diaphragmatic.
   - Mode of breathing
     - Quick; dyspnœa;
     - cardiac apnœa; alteration in rhythm.
2. Palpation
   - Amount of expansion.
   - Fremitus; vocal, bronchial, pleural, cardiac.
3. Mensuration
   - Mechanical measurement of chest.
   - Vital capacity of lungs.

4. Percussion
- Tympanitic sound.
- Dulness or tonelessness.
- Natural resonance.
- High-pitched note.
- Amphoric resonance.
- Cracked pot sound.
- Feeling of resistance.

5. Auscultation
- A. *Respiration.*
  - Natural tracheal sound.
  - Natural bronchial sound.
  - Natural breath murmur; deficient, increased, interrupted; altered rhythm.
  - Larger bronchial tubes—râle muqueux, râle sonore, râle sibilant.
  - Capillary tubes—subcrepitant râle.
  - Air cells—fine crepitation.
  - Tubular breathing over consolidated lung.
  - Cavernous breathing over cavity.
  - Gurgling.
  - Amphoric sounds.
  - Pleuritic rub.
- B. *Voice.*
  - Bronchophony over consolidated lung.
  - Pectoriloquy over cavity.
  - Ægophony.
- C. *Cough.*
  - Amphoric resonance.
  - Metallic tinkling.
  - (Bruit d'airain).
- D. *Succussion.*
  - Splashing sound.

1. INSPECTION

Form of Chest.—You first of all expose the patient's chest, and look at its form. This may at once give you some information as to the general structure of his figure and temperament. Any alteration of the shape of the chest has usually existed from childhood, and may be inherited, but it is seldom congenital. The causes instrumental in producing the change of shape may be accidental, but many of these causes being dependent on the constitution of the parents, may be regarded as indicative of a particular temperament. Abnormal chests may be unusually *large* or *small*. The former are usually acquired at a later period of life, the latter in

infancy. At birth the chest is usually well formed, and its circumference nearly circular; and it is afterwards that alterations take place in its shape. The changes depend mainly upon the state of the lungs within, and also upon the condition of the bones. Anything which prevents due expansion of the lungs necessarily shows its effects on the case which holds them. Any inherent weakness on the part of the infant, or an acquired debility from want of food, might prevent a proper inflation of the lungs, or if these had been once inflated the child might not have sufficient power to keep them expanded, and the lungs would return to the fœtal state or to an "atelectasis" of parts of them. A bronchitis, in like manner, by causing closure of the tubes, might necessitate a collapse of the lower lobes, and thus bring about a non-expansion of the chest.

You can see how, from a want of vigorous inflation, a chest might remain in a contracted condition, and become permanently a long and *narrow* chest. You can also see how, from a bronchitis and want of due inflation of the lower lobes, the sides of the chest might fall in, and in this way be produced a form of thorax hollowed out at the sides, a variety by no means uncommon. If this condition were still further exaggerated, the lower part of the sternum would project and a modification of the pigeon breast would be formed. The true *pigeon* breast would not be produced, I believe, unless the bones were at the same time soft, and therefore this condition not only shows that during infancy there has been a deficiency in the expansive power of the lung, but that the patient is the subject of *rickets*. In rickets the bones are soft, containing less earthy matter than they should do, and instead of it a red gelatinous material; you will observe that when a rickety child is put on the ground, the tibiæ bend forwards, the femurs outwards, and the rami of the pubes are forced inwards to produce the rostrated pelvis. Before these deformities occur, you may sometimes see the chest flattened by pressure against the mother during nursing, and subsequently if the soft state of the bones continue, and anything interfere with the due expansion of the lung, the chest falls in on both sides and the sternum is thrust out in front by the cartilages, like the breast of a bird. At the same time the ribs become bent almost at right angles. This rickety chest is no part, as far as I am aware, of the tuberculous diathesis, and therefore you must not say, when examining the narrow pigeon breast of an adult, that he is prone to consumption. It may be that a narrow chest shows an inherited weakness of constitution as well as

that chest which is flattened at the sides, but neither of these I should regard as the form of chest mostly met with in consumptive persons. Remember, in the first place, that subjects of the tuberculous temperament are well-grown and well-formed people, having the limbs straight and the head and chest large. If there be any peculiarity about the phthisical or tuberculous chest, it is that it is *flattened* in front; that is, broad from side to side, and narrow from before to behind; thus contrasting with the barrel-shaped chest of bronchitis. Not only is it flattened, but the sternum, instead of having any tendency to project, falls in slightly, and is seen to be depressed below the ribs. Whether this formation is one inherited from birth, or acquired during infancy, I cannot say, but I strongly suspect the latter.

I will, therefore, ask you to remember this *flat* tuberculous chest, as well as the *narrow chest*, with the *modified pigeon breast*, in all of which a phthisical condition may be subsequently developed; then, also, *the rickety or pigeon breast*, which is due for the most part to the soft state of the bones.

A *large* chest, or one larger than natural, is acquired at a much later period of life, and is due to long-standing bronchitis or emphysema. This chest is really larger than the natural chest during the most forced inspiration; it is also altered in shape, being rounded or barrel-shaped, the diameter from before to behind being sometimes as long as that from side to side; the back is convex, and the sternum is curved, so that the patient looks as if he were humpbacked; the upper part is rounded and large, whilst the lower is more contracted. If you watch the breathing you will see how it has come about; owing to the bronchitis and emphysema, and consequent difficulty in expelling the air, the diaphragm has pushed up the lungs, and the chest continually heaving, its upper part has become permanently expanded. It is a form of chest worthy of your notice and never to be overlooked, for it contains in its appearance the history of the patient.

Inequality of the two sides.—Now, having examined the chest as a whole, and observed whether it be malformed, look and see whether the two sides are symmetricial; one side may be larger than the other, which may indicate a pleuritic effusion, or a growth, or hydatid in the liver, or one side may be contracted or smaller than the other; this is generally due to an old pleurisy, which has affected the whole side or a part of it; if the upper part of the

chest be contracted, it is very often associated with disease of the lungs, and indicates phthisis.

We next come to the *mode of expansion* of the chest, by observing whether the two sides dilate equally. This is very important to notice, since the impaired mobility of one side necessarily points to recent or old disease on that side; and it is also important to take note of the unequal expansion of parts of the same side, as, for example, of a deficient movement of the upper or lower part, which generally implies the existence of a former pleurisy. You must also observe whether the ribs and diaphragm are doing their work in the right proportion, for there are causes, especially those existing in the nerve centres ruling over the respiratory process, which may either paralyse the movement of the chest or the diaphragm respectively, and therefore we say the breathing is costal or diaphragmatic. Also in cases of emphysema and capillary bronchitis, where the lower lobes of the lungs tend to become airless, a want of due expansion of the chest is very noticeable at its lower part, while the upper part is heaving at every breath.

Mode of Breathing and Rhythm.—You will first observe if the breathing is *quickened*. If so, it may be simply nervous in persons who are well. If associated with acute illness it should make you at once suspect some inflammation in the chest. If the increased rapidity has been constant for some time, you may suspect a chronic disease, as phthisis. *Dyspnœa* is difficulty of breathing, and the term should be used in no other sense. It means the cases where the air enters the lungs with difficulty, owing to impediment in the air passages, and is therefore applicable to cases of laryngitis and bronchitis. A third form of altered breathing is seen in the case of breathlessness, shortness of breath, or *cardiac apnœa*. This is not dyspnœa; there is no difficulty in getting the air into the chest, but the breathless condition is due to the irregular manner in which the blood reaches and passes through the lungs. You must remember that there is an intimate relation between the action of the heart and the respiratory process. The heart beats four or five times to every respiration, and as much blood as is sent in on one side is received on the other. It is very evident, therefore, that to preserve the equilibrium, any increased activity of one organ should be reciprocated by the other, and this occurs in active exercise or in febrile conditions, but in any disease of the lungs inducing impediment to the flow of blood, the heart suffers; it becomes overloaded, the venous system gorged, and serum at last exudes in the

form of dropsy. In like manner if the heart be acting irregularly, or be weak, and therefore cannot propel the normal amount of blood into the lungs in a regular manner, or cannot receive it in due quantity on the other side to propel it onwards, the respiratory process is interfered with, the due relations between the circulating blood and air is deranged, and the patient feels distressed or breathless. Of course the lung may be gorged at the later periods of heart disease, and a real difficulty of breathing ensue, but at earlier periods the condition is one of breathlessness or apnœa. The proportion between the respiratory act and the beat of the heart is as 1 to 4½, since the number of expirations in a minute is 16, and beats of the heart 72.

The *Rhythm* also may be altered. It may surprise you when I say that very different opinions have been held as to the exact mode and time in which expansion of the chest takes place during breathing, but it is so, and the fact is owing to the difficulty we experience in attempting to measure accurately the movements of the chest, since directly an attempt is made to do so, the subject of the experiment having his attention fixed on himself, the natural process is thrown out of gear. The graphic method has lately been used, but this is also open to the objection named. You may remember this fact as near the truth—that if the time of one respiratory act be divided into ten parts, five would be consumed in inspiration, four in expiration, and one in rest. I am speaking of the movements only; if you regard the breath sounds heard by the ear, you will find that there is but one continuous or single respiratory murmur. In ordinary quiet breathing this is heard only during inspiration, no sound being heard whatever during expiration. In forced breathing some amount of murmur may be noticed at the commencement of expiration, but if so it is quite continuous, and not to be dissevered from the sound heard during inspiration. If, then, sounds are heard during expiration they are abnormal and denote disease. They are produced in the bronchial tubes, and therefore are not normal sounds occurring at an unusual period, owing simply to an alteration in the rhythmical movements of the chest, but they are really new and morbid sounds. Expiratory sounds, remember, are always abnormal, both in character and rhythm.

2. PALPATION

We now come to the next mode of examining the chest, and that is by laying the hand upon it, or by palpation. We have two objects in view by this procedure; first, to ascertain the *amount* of *movement* in the chest, and, second, the absence or presence of *fremitus*. As regards the first, the hand will often distinguish what the eye cannot; of course in well-marked cases of disease, such as pleurisy, especially with effusion, the non-expansile power of the chest is readily discernible, but occasionally there are mild cases of pneumonia or broncho-pneumonia, especially in old people, where the ear can detect nothing, but the hand can readily appreciate the difference of expansion on the two sides. I have ascertained the existence of pleurisy in a patient lying insensible by this means.

Then, again, we lay the hand on the chest for another important reason, and this is to ascertain the modification of the *vocal fremitus*. This is a most important physical sign, but constantly overlooked by students. In health, when we speak, the voice vibrates through the chest, and this thrill is felt when the hand is placed upon it. Now, in some cases, as in hepatisation of the lungs, this vocal thrill or fremitus is very commonly intensified, whilst in effusion of fluid it is altogether absent. The hand will not only appreciate the difference in vocal thrill, but it will often detect a pleuritic fremitus in acute pleurisy or a rhonchial fremitus in bronchitis or a thrill in cardiac disease, more especially in stenosis of the mitral valve.

3. MENSURATION

Under this head we include the actual measurement of the *size* of the chest by means of tapes and the measurement of its *movements*. We include, thirdly, a measurement of the air which can be breathed into the lungs, and which is also called the *vital capacity*. As regards the first, I will refer you to your books, where you will see various kinds of apparatus used for the purposes of measurement; some of these taking the form of the chest, and others which are graduated, enabling you to compare the size of the two sides. There are also instruments which, by being placed on the chest, move the

hand of a dial, and thus enable you to measure the amount of expansion.

The *vital capacity*, or amount of air breathed, is taken by means of a spirometer, and I will here remind you of what you have already learned on this subject, because the facts are applicable to cases of disease. You know that when you breathe, the lung undergoes only a moderate amount of expansion and contraction; when you have expired to the utmost the chest is still full of air-containing lung; just as a river still contains water when the tide has ebbed. This air, which corresponds to the difference in amount between that in the inspiratory lung and that in the expiratory lung, is called the *tidal* air. The air which remains in the lung after the most forced expiration is styled the *residual* air. Now, in ordinary inspiration we do not inflate the chest to the very utmost, but, if we do, we breathe in an additional amount of air, and this in forced inspiration is called *supplementary*. Such a condition occurs when one lung is diseased or compressed, and additional work is thrown upon the other, and it is for this reason therefore very important to notice. You may also remember that when in the dead body you open the chest, the lung collapses, but if it be taken out and examined, it will be found still to contain air, and it floats in water; this air is called the *persistent* air. In some cases, however, as in pleuritic effusion, every particle of air may be squeezed out of the lung; it is then a fleshy mass, and is said to be carnified. The amount of respirable air is the difference between that in the fully expanded lung and that in the most forced expiration—or the tidal and supplementary air together. The spirometer has been found of little use in practice, owing to the difficulty there is in persons understanding how to use it, and then again because the amount of expansion depends upon other circumstances besides the size of the chest; as, for instance, the muscular strength of the patient; therefore, a person used to athletic exercises would show a higher range on the instrument than one whose chest was of equal dimension, but who was fat and had led a sedentary life.

4. PERCUSSION

This is by far the most difficult part of physical diagnosis, because there is no universal agreement as to the true conditions which produce the various sounds, and there is no exact definition

given to the terms which are used; moreover, many writers adopt expressions of their own selection which cannot be accurately interpreted by the reader. For instance, the common term "resonance," how impossible it is to define it or convey in any words to you what I understand by it; and the same, though to a less extent, with the word "dull;" but when we read in books of such expressions as "empty" and "full," the words often convey no meaning at all. I do not find fault with the attempt to give names, for it is evident that, under varying conditions, different sounds must be elicited by striking the chest; but since, at the present time, we are not all agreed as to the true causes producing them, it is impossible to use expressions which are intelligible to all. I say there is no general agreement as to how the sounds are produced, some maintaining with Piorry (who has written a poem on percussion) that every part of the body gives out its own note on percussion, whilst others, with Bennett, have been content to teach that there are three main sounds according as we percuss over a solid, a liquid, or air; these would be illustrated by striking over the liver, bladder, or stomach, and therefore might be styled respectively parenchymatous, humoral, and tympanitic sounds. Without systematizing too much, we are generally content to say that we elicit from the walls of the chest a given sound which varies with the condition of the ribs and their covering, but more especially according to the nature of the contents within. The sound is really produced by a vibration of the ribs which can be felt by the finger, and the note varies more especially with the contents. I have no doubt that the subject might be taken up advantageously as a simple question of acoustics, and appropriate terms be given. You will see, on a moment's thought, that the nature of the object struck must determine a difference in the note, as also whether it be solid or hollow; the sound again varying with the character of the contents.

When one considers that percussion of an object is a simple physical method of ascertaining what is within, it is remarkable that its practice has been left to modern times; we know if we knock at the door of an empty room that the hollow sound produced will tell us of its condition, just as we know by striking a barrel whether it is empty or full; in the same way the builder knows by striking a wall whether it be composed of bricks or lath and plaster; and when we are stopping on our journey at the railway station, a man comes and knocks the wheels in order to discover a flaw. One of the most

remarkable illustrations of the information to be obtained by percussion occurs in 'The Mystery of Edwin Drood,' the last novel which Dickens had in hand when death suddenly disarmed him of his pen. It would be most interesting to know whether he gained the idea from observing the methods used amongst us medical men, or whether, quite irrespective of us, he had merely in a fanciful and somewhat exaggerated manner described what he had actually seen performed amongst workmen.* The passage is sufficiently interesting to read to you, if only to show how percussion is merely a matter of physics.

"Is there anything new down in the crypt, Durdles?" asks John Jasper.

"There's an old 'un under the seventh pillar on the left as you go down the broken steps of the little underground chapel. Now, lookee here. You pitch your note, don't you, Mr Jasper?"

"Yes."

"So I sound for mine. I take my hammer and I tap. (Here he strikes the pavement). I tap, tap, tap. Solid! I go on tapping. Solid still! Tap again. Holloa! Hollow. Tap again, perseveringly. Solid in hollow! Tap, tap, tap, to try it better. Solid in hollow; and inside solid, hollow again! There you are! Old 'un crumbled away in stone coffin, in vault."

"Astonishing!"

To simplify the matter as much as possible, I shall merely describe to you the *four* most striking different conditions in which the chest may be placed in health and disease, and which will correspond in the main to the solid, liquid, and gaseous divisions already mentioned. The chest may be full of *air* or full of *liquid*, or may contain a *solid hepatized lung*, or, fourthly, the *natural lung*. Now, the sounds elicited by striking the chest under these different circumstances can be imitated by taking four boxes, and filling them respectively with *air*, *water*, *wood*, or *wool*. Or, if we took an empty thorax and filled the one side with air and the other with water, we should have two very different sounds produced upon striking them. If we percussed the one side it would sound like a drum, so that we should feel no doubt that it was hollow, and might call the sound *tympanitic*. Now, if we struck the other side we should elicit no sound whatever, no vibration would occur in the ribs, and there-

* Since this was written, Mr. John Forster, the author of the 'Life of Dickens,' informs me that he has no doubt the novelist wrote from his own keen observation of the methods adopted by workpeople.

fore no note. This side would be said to be *dull* on percussion, although a better term than dulness would be *tonelessness;* it would be as toneless as striking the thigh. Now, if we took another chest and placed within it some wool or spongy lung, we should have necessarily an intermediate sound; we should not have the hollow sound of simple air, nor the total want of vibration, as in the case of fluid, but another and altogether different note would be emitted. This note, produced by striking over a healthy lung, is called *natural resonance.* I cannot describe it to you in so many words, but you must familiarise your ears to the note, and then you will take it as a standard; any sound above it is called hyperresonant, as you have in an emphysematous lung, and an excess of this again is the tympanitic note, which is composed of a few vibrations of great amplitude; whilst any sound below it until perfect dulness is reached implies the existence of more solid material within.

Now, the greatest difficulty in the explanation of a note exists in the case where a solid fills the chest, as when a piece of wood is introduced within it, or when, as in the living subject, the lung is hepatized. You will see that dulness corresponds to tonelessness, and this very often means the non-vibratile condition of the walls from the presence of solid matter within the chest; but solids do not necessarily cause an absence of tone. Thus, it is true that, if I strike over the liver or a chest full of fluid, I have what is called a dull sound, but if I strike over the sternum or head a different note is elicited. In the latter case it shows that the bone itself produces a sound, but even then the solids and fluids produce different notes, as can be seen by striking a barrel full of fluid, and a solid block of wood of the same size and shape. The latter vibrates, but the vibrations are shorter than in the hollow barrel, and not so enduring, and a *high-pitched* note is produced. This, then, is the term for a note which we often meet with in striking over a solid organ or a hepatized lung.

The vibration and non-vibration of the chest do not necessarily indicate the presence of air or liquid beneath, for, in the case of the barrel, if a thick piece of paper were spread over its inner surface, it would not vibrate on slight percussion, and it might consequently be thought that the vessel was full. An analogous condition occurs in the human chest when a thick layer of pleura lines its inner surface. This prevents vibration when the chest is percussed, and the impression conveyed to you might be that of fluid in the chest, and consequently it has happened that persons have been tapped when they

have had no other disease than a thickened pleura. There is also the converse fact that a vessel may be so full of air and tightly stretched that it will not vibrate, and thus the hollow sound does not appear; this may occur in pneumo-thorax, and is sometimes met with in extreme tympanites of the belly, where, from the great stretching of the stomach and intestines, no tympanitic sound is elicited. The experiment may be artificially made by blowing out a bladder, which is resonant or tympanitic until stretched to the utmost, when the tympanitic sound is lost.

I shall leave you with these four conditions of the chest—viz., where it is full of *air*, *fluid*, a *solid*, or *spongy lung*, as the most important, and let you learn up minor conditions afterwards. I will, however, speak of two other sounds, which it is necessary to remember. I told you that when you struck over a hollow space you had a tympanitic sound, but this term is applicable only to the drummy state produced by a very large collection of air. If the hollow be circumscribed as in a cavity in the lung, we adopt the term *amphoric*, which is a sound resembling what is produced by flipping the cheeks when stretched to the utmost, or a hollow india-rubber ball. An important modification of this is the *cracked-pot sound*, produced under the same circumstances as before, but with the addition of a hole in the cavity communicating with the external air. Thus, it would be produced in the india-rubber ball, with a hole in its walls, or it can be produced in the cheek by slightly separating the lips, or still better by striking the closed hands on the knee—the air rushing out between the fingers producing an almost metallic ring. In the case of a cavity, we request the patient to open his mouth, and then if there be a bronchial tube leading from the cavity direct to the trachea, and we strike sharply over the cavity, a well-marked cracked-pot sound is produced. This is almost pathognomonic of the existence of a vomica, for it is seldom heard under any circumstances, although on one or two occasions, in cases of hepatization of the upper lobe, I have heard a sound somewhat resembling it. In children also with elastic chests, it is easy to produce it by striking the walls whilst they are crying.

Under the head of percussion, I might refer to another physical condition, which becomes apparent during the act of striking; I mean, the sense of *resistance*. When you percuss over a resonant or dull chest, your finger appreciates the vibration, or the want of it, and not only this, but it is aware whether a spongy or hard substance is beneath. In the latter case there is not the slightest

giving way of the ribs, and the finger appreciates, by a sense of resistance, that there is a solid beneath.

Now, it is customary to divide the chest into regions, and if you look into your books you will see various ways in which this is done. I think myself it is useless to get up accurately any scheme of the kind, as the terms are always ready at hand when we require them, as infraclavicular, suprascapular regions, and so on. You will find that the front part of the chest is more resonant than the back part, and of the latter that the lower portions are more resonant than the upper part covered by the scapula and fleshy muscles.

By percussion on the right side you will find the lower part dull from the presence of the liver, the lungs and liver varying their position during respiration; thus, during inspiration the lung reaches to the seventh rib, and in expiration to the fifth rib, the medium of course being the sixth; we, therefore, generally say that the resonance reaches on the right side as low as the sixth rib; but there is no sharp line between the resonance and dulness, owing to the lung overlapping the liver, and therefore the two sounds gradually merge into one another. On the left side the lung only reaches to the fourth rib, as it there turns off over the base of the heart.

5. AUSCULTATION

An explanation of the sounds heard by applying the ear to the chest is far more easy than that of the percussion sounds; indeed, I believe, if a novice were to apply his ear to the lungs in various states of disease, he might at once infer what the physical conditions were within the chest which produced them, such as the rattling of bubbles, the hollow sound of a cavity, the meaning of negative signs in pleuritic effusion, &c. I shall merely describe to you the most important auscultatory signs, and those which it is necessary for you to retain in your memories; you can afterwards learn the subject more fully either in practice or from your books.

First, as regards the natural sounds. If you place your ear near the trachea you will be aware that the air is passing in and out of a large tube, and if you place your ear over the shoulders you will hear the same sound in a lesser degree. This is called *tracheal* and *bronchial* breathing. If, now, you place your ear over any part of the chest you hear the ordinary breath sound or *respiratory murmur*. This has been likened to various familiar noises—as the rushing of

the air through the foliage of a thick forest, or the murmuring of the waves on the sea-shore, but I think no closer resemblance to it can be found than that which is produced by gently blowing through the lips. This breath sound is one and continuous. In ordinary quiet respirations it is heard only with inspiration. In forced breathing a sound may be heard at the commencement of expiration, but if so it is added on to the inspiratory sound so as to be one and continuous with it. As the chest falls, as a rule, no sound is heard; if there be any expiratory sound audible, it is abnormal and signifies disease. Much controversy has taken place as to the seat and cause of the respiratory murmur. It is clearly not due simply to the exit and entrance of air, seeing that the cells of the lung do not take in and expel air at every respiration; it has therefore been thought due to the propagation of sound carried down from the larynx; but then, again, experiments on animals by opening the windpipe have disproved this. If due to the impingement of air upon any part of the air passages, the seat of this must be in the ramifications of the smaller bronchial tubes; but it may be caused simply by the unfolding of the lung as the diffusion of gases takes place.

As regards the modification of the breath sound. You may remember that it may be altogether *deficient*, showing consolidated lung or compressed lung. It may be *exaggerated*, owing to a portion of a lung doing increased work to compensate for a deficiency in another part. This exaggerated breathing is called also *supplemental* or *puerile*, because it resembles the loud breathing heard in the lungs of children. Besides exaggeration and actual deficiency it may be altered in rhythm, and a sound be heard with expiration as well as inspiration; also, owing to a deposit in the lung, as occurs in an early tubercular disease of the apex, the respiration may be *jerking* or saccadé.

Now, as regards the *stethoscope*. You must not be led to believe that this is an instrument whose use is to be cultivated by practice, like a violin, for it is merely a handy method of applying the ear to the chest. There are many physicians who prefer their naked ear to the stethoscope, and there are occasions when it is preferable. I think by the naked ear laid on the chest we may obtain a more general idea of the condition of the lungs, whilst by means of the stethoscope we circumscribe and locate the sound. I need not tell you that it is not an instrument framed to intensify sounds, or render others audible, which would not otherwise have been per-

ceived, and yet sometimes I should think, from the observations of students about learning the use of the stethoscope, that they had some notions of this kind. You have to hear certain sounds with your ear, or by means of the stethoscope, and then interpret them. I shall not, therefore, waste your time in displaying before you a hundred varieties of stethoscope and dilate on the advantages of each; but give you some general idea of the instrument, and relate to you the history of its invention. You will then see the principle of its construction. Laennec says, "In 1816 I was consulted by a young woman labouring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail, on account of the great degree of fatness. The other method—percussion—just mentioned being rendered inadmissible by the age and size of the patient, I happened to recollect a simple and well-known fact in acoustics, and fancied at the same time that it might be turned to some use on the present occasion. The fact I allude to is the augmented impression of sound when conveyed through certain solid bodies, as when we hear the scratch of a pin at one end of a piece of wood on applying our ear to the other. Immediately on this suggestion I rolled a piece of paper into a kind of cylinder, and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than ever I had been able to do by the immediate application of the ear." "I forthwith commenced a series of experiments at the hospital, Necker, which have been continued to the present time. The first instrument which I used was a cylinder of paper, formed of three quires, compactly rolled together, and kept in shape by paste. The longitudinal aperture, which is always left in the centre of paper thus rolled, led accidentally in my hands to an important discovery. This aperture is essential to the exploration of the voice. A cylinder without any aperture is best for the exploration of the heart; the same kind of instrument will, indeed, suffice for the respiration and rattle, but both these are more distinctly perceived by means of a cylinder which is perforated throughout, and excavated into somewhat of a funnel shape, at one of its extremities, to the depth af an inch and a half." "In consequence of various experiments I now employ a cylinder of wood an inch and a half in diameter and a foot long, perforated longitudinally by a bore three lines wide, and hollowed out into a funnel shape to the depth of an inch and a half at one of

its extremities. It is divided into two portions, partly for the convenience of carriage, and partly to permit its being used, of half the usual length. The instrument in this form—that is, with the funnel-shaped extremity—is used in exploring the respiration and rattle; when applied to the exploration of the heart and voice, it is converted into a simple tube with thick sides by inserting into its excavated extremity a stopper or plug traversed by a small aperture, and accurately adjusted to the excavation. This instrument I have denominated the *stethoscope*."

You will see this original instrument in the studies of many old practitioners. By fashioning its sides and enlarging the ear-piece and trumpet-end the present stethoscope has been evolved. Laennec was mistaken in supposing that the central aperture was necessary for the perfection of the instrument, for many medical men carry solid stethoscopes, showing that it is the solid wall which conveys the vibration of sound. The conduction is probably facilitated by the central perforation.

Now, beginning with the abnormal sounds, we will first take the air passages and divide them into the *larger tubes*, the smaller or *capillary tubes*, and the *air cells* or alveoli. First, as regards the larger tubes and the abnormal sounds produced therein, I will ask you to remember the three sounds given by Laennec, as these are the most important. He observed that when mucus was contained in the tubes, as in bronchitis, and the air passed through it, bubbles were produced, and he recognised these as the same sounds which are often heard during the moments of dying, or the so-called death rattles; he, therefore, called this sound "*râle muqueux*," or, technically, rhonchus. He then, also, observed that in bronchitis there might be other sounds when the disease was passing off, or in cases where the disease was chronic. These snoring and whistling sounds he called, the one "*râle sonore*," and the other "*râle sibilant*." The former is heard in the larger tubes, more especially during expiration, and is a low-pitched grave note, whilst the latter is heard mostly with inspiration in the smaller tubes, and is a high-pitched note. These may sometimes be accompanied by a fremitus felt by the hand.

Next we come to the *smaller tubes*, which are affected in capillary bronchitis. The air passing through the mucus during inspiration and expiration, produces a double râle; it is evident to the ear that the bubbles are much smaller than those produced in the larger tubes, and that a larger number are heard over a given space. If this

should be at the usual seat of capillary bronchitis, at the lower part of the lungs, the diagnostic significance of this rhonchus cannot be mistaken. Since the sound produced in the air cells is called a crepitant râle, this one produced in the capillary tubes is designated the *subcrepitant râle.* Remember, the sound is very fine and a double one.

The *crepitant râle* or *fine crepitation* is the sound heard in pneumonia. The crackling is very minute, so that a great deal of it is heard under a very small space of the stethoscope. There is no better similitude than that usually given of rubbing together of a lock of hair near the ear. It is a simple continuous sound, and in this way it is distinguished from the subcrepitant râle before mentioned. It has not positively been decided in what manner the sound is produced, the same questions and difficulties existing as in the case of the natural respiratory murmur. As air does not directly enter the alveoli of the lungs and again immediately escape, so the sound cannot be caused by the simple passage of air; but just as the natural sound is probably caused by the unfolding of the healthy lung, so in like manner is the crepitation in pneumonia produced, when viscid secretion is present. This would, of course, replace the natural breath sound by a crackling.

We now pass to other morbid conditions of the lung. If a lung be consolidated, as in pneumonia, the natural breath sound is obstructed, and nothing is heard but the air passing in and out of the tubes. This sound is carried direct to the ear through the good conducting solid lung, and thus we have what is called *bronchial or tubular breathing.* We, in fact, hear all over the lung the same sound, which is a normal one, when heard over the bronchi between the scapulæ. As the lung cannot expand, it has been thought that no air can pass in and out of the lung, but that the sound which is heard is only transmitted from the healthy side. There seems no reason, however, to doubt the capability of air being dragged in and out by the current which passes down the trachea to the healthy lung of the opposite side.

Now, if there be a large hole or cavity in the lung, the air is heard to pass in and out through the tube which enters it, and the ear can at once discern from the sound that a cavity must be present. It is therefore called *cavernous* breathing. The sound is with difficulty distinguished from the tubular before mentioned, and I shall refer to this again. If fluid be present in the cavity, and the air passes through it, we have the same sound as mucous râle in bronchitis,

but being localised we give to it the name of *gurgling*. When we use this term we imply the existence of a cavity, but in many cases the sound not being distinguishable from a mucous râle a bronchitis may really be present.

Sometimes the cavity in the lung is of great size, occupying half the organ, and the walls at the same time may be very thin and large tubes may be entering into it. Under these circumstances the ear at once appreciates, from the resounding of the voice and cough, and from the excessive hollowness of the sound as the air enters, that the cavity is very large; indeed, the breath sound is like that produced by blowing into a pitcher; it is therefore called *amphoric* breathing. The sound is more commonly heard and to greater perfection when the space full of air is altogether outside the lung, and in the cavity of the pleura. Then it is that these amphoric sounds are best heard.

Having now told you of the morbid sounds which you hear in the tubes and lung substance whilst the patient breathes, there is only one other structure to remember, and that is the pleura. If lymph be on the surface of the lung, and the patient breathes, you may hear a *pleuritic rub*. This is generally double, but it may be heard with inspiration only. It is heard always best at the lower part of the chest where the lungs are most movable.

Voice.—Having endeavoured to discover the various morbid sounds during inspiration, we now make the patient speak. We ask him questions or tell him to count. In health, when a patient speaks, the voice vibrates through the chest. The words do not remain distinct, but are confused and lost. In disease, however, the articulation may be precise and defined, the words passing directly into the ear; in fact, more clearly than the words spoken by the mouth pass to the other ear through the air. Now, in the case of a cavity, as I mentioned before in speaking of Laennec's discovery of the stethoscope, the voice becomes painfully distinct to the ear. This is called *pectoriloquy*. The intensity often varies with the tone or timbre of the voice, so that sometimes a whisper is carried more directly through the chest to the ear than would a full-toned note. It has been said that if a patient blows the scale through pitch-pipes some notes are more easily transmitted than others. Now, there are other circumstances under which the voice comes direct to the ear in the same manner, and that is when the lung is consolidated, as in pneumonia. The voice is transmitted down the bronchial tube, and carried through the good-conducting lung direct to the ear, as before

mentioned. Under these altered conditions the sound is not called pectoriloquy, but *bronchophony*. The usual explanation of the better conducting power of hepatized lung to account for the phenomena is to me quite sufficient, and therefore I teach it; but there are those who are not satisfied with it, and maintain that bronchial breathing and bronchophony are produced for other reasons; they say that the voice is produced in the larynx, and that the bronchial tube being surrounded by hardened walls takes up the note produced in the larynx above and repeats it; that the tube, indeed, consonates with the original sound, reproduces it, and that it is this new sound which is conveyed to the ear. Bronchophony is not, therefore, due to the better-conducting properties of the consolidated lung, but from the tube consonating with the larynx and its sounds above. I mention the theory because you are sometimes asked to explain bronchophony on the theory of consonance. In whatever manner it may be caused, a much more important question is, how are we to distinguish cavernous breathing from tubular breathing, and pectoriloquy from bronchophony? Now, I may as well tell you at once that some of the best authorities on auscultation deny the possibility of our doing so, and I am of this impression, because I have seen distinguished men, and those who would not admit their incapability of diagnosing a cavity, fall into the error named. The point is this—that a portion of lung containing a cavity is productive of the same sounds as a portion of consolidated lung with a moderate-sized tube passing through it. In both there is a hollow sound during respiration, and in both the voice is directly transmitted to the ear during speaking. In most cases no difficulty exists in diagnosing the cause, since we have other circumstances to guide us; for example, if a patient has had for a day or two all the other signs of pneumonia, and we find, on examining the lower lobe, the physical signs just indicated, we say he has bronchial breathing and broncophony, implying thereby a consolidation of lung. If, on the other hand, we have a patient long ill with all the symptoms of phthisis, and on examining the apex we meet with the same physical signs, we say he has cavernous breathing and pectoriloquy. The question remains—do these sounds, which depend on different causes, and to which we have given different designations, in any way differ in character? I think they do not, for if the accompanying circumstances before named are not present to assist us, but, on the contrary, these circumstances are reversed, we are prone at once to fall into error. Should our phthisical patient get a

sudden consolidation of the upper lobe a cavity would be diagnosed, and at the lower part of the lung the before-mentioned physical signs would rarely be interpreted otherwise than by consolidation. Even if we are right in the diagnosis of a cavity in the apex, we are not sure whether it be one of a large size or a smaller one with hardened walls; the conducting powers of the latter being equivalent to larger dimensions of the vomica. Thus it is that so much controversy has taken place respecting the healing and cicatrization of cavities in the lung; the observers never agreeing as to the positive existence of a cavity—since the physical signs would be much the same in the case of a consolidated apex with a tube passing through it.

I have dwelt longer on this subject than on any other, for in the whole range of auscultation it is by far the most important and difficult one to unravel.

Ægophony.—This is the sound produced by the passage of the voice through a thin layer of fluid; in the case of pleuritic effusion, lymph is thrown out, which may be of varied consistence, and form a film over the lung; if under these circumstances the ear be placed over the affected spot, and the patient made to speak, the voice vibrates in a very high key, and is therefore called the goat's voice or "punch's voice." If there be other circumstances which convey the voice direct to the ear, as in hepatization of the lung, the peculiarity of sound is still more decided, and therefore it is that in pleuro-pneumonia œgophony is best heard, and is then styled *bronchophonic œgophony.* Even in simple pleurisy the fluid may be sufficient to compress and to a certain extent densify the lung, and thus tend to produce a bronchophonic sound.

Cough.—Before we remove the ear from the chest, we often request the patient to cough, as we may learn much from the sound or the effort made to produce it. We might first observe that all those conditions which tend to carry the voice-sound more distinctly, would be operative in the same manner in the case of the cough, but the effects produced are useful in other ways, as by dislodging mucus, and thus allowing air to pass into the lung or into the cavity. In the latter case we may sometimes place our ear over the cavity, and though the patient breathes tolerably forcibly, yet we hear no sound, but immediately he coughs, air is driven into the space, cavernous sounds are heard, or, if fluid be present, gurgling also. In the case of large cavities it is absolutely essential that the patient should cough in order to drive air into the space, and so

produce the characteristic signs. If air be present in the chest, as in pneumo-thorax, and the patient coughs, a metallic ringing sound is heard through the chest or *amphoric resonance*, as it is called; if fluid be disturbed, drops of it fall down, and the sound produced is a high and metallic one, technically called *metallic tinkling*.

I might also mention here the remarkable metallic sound produced in these cases by artificially striking the chest instead of making the patient cough, although this method would more correctly come under the head "percussion." If you have a case of pneumo-thorax and lay your ear upon the chest, and then another person place a coin on the surface and strike it, you will hear the sound reverberate through the chest like a deep-toned church bell. This was called by the French *bruit d'airain*.

The method is of great use in those cases where the opening in the lung has closed, so that no metallic or amphoric sounds can be produced by breathing or coughing.

If fluid and air be together in the chest, we have the case which is styled *pleuro-pneumo-thorax*, the fluid being in the most dependent part and the air above. The chest under these circumstances is like a vessel half-full of fluid, and therefore allows its contents to splash about in it if shaken. If the ear be placed on its surface and the patient shaken, a distinct splash can be heard as the fluid strikes the sides. This physical sign was known to Hippocrates, and, therefore, this mode of physical examination is sometimes called "Hippocratic succussion."

PNEUMONIA

In the whole history of medicine no disease has attracted so much attention as pneumonia. The acuteness, severity, and remarkable nature of the complaint have given to it this prominent position, and for these reasons, like the sacrificial frog, it has been made the subject of every experiment in therapeutics. It is still regarded as the typical affection in which we are to try all the new antiphlogistics, including digitalis, aconite, phosphorus, veratria, and a host of other remedies.

If you consider the remarkable nature of the complaint, you will almost anticipate me in your knowledge of what the symptoms and physical signs must be. Think for a moment of a spongy organ like the lung, destined to contain air, becoming in a few hours quite solid from the pouring out into its tissue of a pound or two of albu-

minous substance. You might know what a physical examination would show, and you would be prepared for the distress of breathing and high state of fever. Since, then, the symptoms depend so entirely upon this altered condition of the lung, it will be advisable to describe in the first place the anatomical changes which the organ undergoes during the process of inflammation. You will find that ordinary pneumonia involving a whole lung or a large part, is at the present day often styled "croupous," and this I will explain. You know that the lung tissue and the bronchial tubes are so anatomically and physiologically different in the adult, that the inflammation of the two structures remains totally distinct, pneumonia being one thing and bronchitis another. In children, however, it is not unusual for a bronchitis to continue downwards into the lobules of the lungs, and so a pneumonia is set up which has a different origin from that in the adult, and is purely lobular in its character. This is sometimes met with in adults, but, if so, it is a chronic disease. We have, therefore, a *lobar pneumonia* and a *lobular broncho-pneumonia*. Now, it is said that the character of the inflammation is different in the two cases, in the same way as it differs often on the same mucous membrane, as, for example, in the larynx and trachea, where we meet with a croupous inflammation and a catarrhal inflammation; in the one case the surface is covered with a fibrinous membrane, and in the other with a corpuscular mucous secretion. Now, it is maintained that in ordinary lobular pneumonia, the inflammatory product partakes of the nature of the first, and in broncho-pneumonia, or lobular pneumonia, the inflammatory product resembles the second; the former has, therefore, been styled *croupous* pneumonia, and the latter *catarrhal* pneumonia. I explain all this because the terms are getting into use, but at present withhold my opinion as to the correctness of the statements with regard to the histological process.

Anatomical changes in lobar pneumonia.—It was long taught that inflammation of the lungs consists in the exudation of a lymph into the interstitial substances or parenchyma of the lungs, but of late years the doctrine which was always taught by Addison in this school, and who was the first to demonstrate the true nature of pneumonia, is now universally adopted. It is,—that the air-vesicles or the alveoli constituting the dilated termination of the bronchi contain the inflammatory products. Addison was led to his conclusion forty years ago by actual observations as to the seat of the exudation by the structure of the pulmonary cells and by the

proved absence of any supposed intercellular spaces into which lymph could be effused.

The changes which take place in the lungs are of the same kind as are witnessed in other structures, viz., first, an intense hyperæmia followed by a fluid and then by a solid exudation. The change, therefore, which the organ undergoes is a most striking one, being none other than the conversion of a light spongy organ, normally full of air, into a heavy solid mass, and all within the space of a few hours. The different stages of the process are sufficiently well marked, and are traceable during life by various physical signs, which accord exactly with the changed conditions of the lung. They resemble what is seen in other structures during inflammation, viz., hyperæmia and vascular engorgement followed by an exudation.

The first marked change observable in the lung after death is that of *engorgement with serous exudation*, and this is usually called the first stage of pneumonia. On taking the organ out of the chest it is found to be more bulky and heavy than the healthy lung; it pits on pressure, and when cut through it is much redder from sanguineous engorgement; a quantity of serum pours from it, and, owing to the lung still containing air, the serum is frothy, and protions of tissue still float when cut off and thrown into water; if now the finger be forced into the lung it will be found to be readily lacerable, and this is a means of distinguishing it from simple œdema or dropsy of the lung. This softening is one of the best signs of inflammation, and it further shows that the fibrous tissue which forms the walls of the pulmonary air-vesicles is more or less involved in the inflammatory change, for although we give up the old theory of an interstitial inflammation, yet we must hold that, in connection with the cell-formation which takes place, the texture of the lung is loosened by an exudation into its meshes, in the same way as in inflammation of other structures; as, for example, of the peritoneum, where it may be observed that, although the exudation takes place from the surface of the serous membrane, yet the coats of the intestine are infiltrated with lymph, and are readily separable from one another. It is during this stage of serous exudation in the lung that fine crepitation is heard by the ear.

It is not usual until the lung has reached this stage in the inflammatory process that inflammation is recognisable during life, and it is rare for it to be seen earlier on the post-mortem table. Yet exceptionally this is the case, and it must be evident that prior

to the exudation of any fluid there is a stage of simple engorgement. The air-vesicles would be encroached upon by the distended blood-vessels causing a harsh and deficient breath-sound. This early condition has been recognised by Stokes, and confirmed by Addison, so that it would more justly be styled the first stage of pneumonia, and that to which this name is given would then constitute the second stage. I shall adhere, however, to the usual nomenclature.

The second stage of pneumonia, that of red hepatization.—As in the course of inflammatory effusions in other parts of the body the serous exudation at first poured out becomes more solid, so the same occurs in the lungs, the serum makes way for lymph, until the air-cells become completely blocked by it. The lung at this time when cut through no longer exudes serum, but a solid matter squeezed from the cells; the blood vessels, however, are still gorged with blood, which is in part stagnant within them; at the same time the secretion which has been poured out contains some blood-globules and hæmatine, and thus the organ still retains its red colour, though not so bright as in the healthy state. This dull red solid lung resembles, under these circumstances, a piece of liver, and thus the term hepatization has been given to it. On examining the cut surface it is seen to be slightly granular from the projection of the distended air-vesicles; it no longer emits any fluid or air on pressure, but rapidly sinks in water. It is also very lacerable when the finger is thrust into it.

The third stage, that of grey hepatization.—This is a further development of the previous stage by a more complete solidification. The air-vesicles are more tightly filled with inflammatory matter, the cut surface is still granular, sinking in water and easily lacerable; but the exudation contains less colouring matter, and the blood has been squeezed out of the capillaries, and thus the whole colour of the lung is paler. The grey colour, indeed, which the lung presents is due mainly to the natural pigment which it contains, and thus it happens that a hepatized lung is much darker in aged persons, where more natural pigment exists, than in children, where pigment is almost deficient; it follows that the hepatized lung of children is white or yellowish-white. The nature of hepatized lung can be readily understood if you can suppose the minute bronchial twigs and air-cells tightly filled with albuminous material, and, indeeed, it very much resembles the preparations in our museum where the organ is injected with tallow. When a

lung is solidified or hepatized in the manner spoken of, it is seen filling the chest when the body is opened, instead of collapsing as a healthy lung should do; it forms, indeed, a mould of the thorax, and when removed the form of the ribs may be seen impressed upon it. Not uncommonly a slight pleurisy has accompanied the pneumonia, and then a delicate layer of lymph may be found on the surface of the lung and easily separable from it. A lesser amount of pleurisy is very commonly present where the membrane is seen to be opaque whitish, or has lost its lustre. This is so common that it is only when a considerable amount of lymph has exuded from the surface, that the name pleuro-pneumonia is applicable. In comparing the solidified with the healthy lung, it will be found that in some cases as much as three pounds of solid matter must have been rapidly exuded into the tissue. During life, in the second stage of pneumonia, there is an absence of all natural breath-sound, but in its place there is bronchial breathing and bronchophony with dulness on percussion.

Now, a few words as to the minuter changes which are going on in the lung, and the formation of the inflammatory products which are composed of cells held together by a fibrinous material, occupying the air vesicles. These are changes probably of the same kind which are in progress during the course of inflammation elsewhere. Before microscopic times, pneumonia was regarded as an exudation of lymph into a supposed parenchyma of the lung, but when the organic nature of an inflammatory product was discovered the theory of Schwann was applied, and it was taught that the cells sprang up in a cytoblastema, poured out from the congested blood-vessels. Afterwards came Virchow's theory, which was also made applicable to the lungs, and so pneumonia was nothing more than a proliferation of the endothelial cells of the aveoli. More lately, since Cohnheim's views have come into vogue, it may be supposed that a part of the new exudative material may be formed from the migrated white cells of the blood. The term croupous implies that the exudation in the alveoli is composed mainly of a fibrinous liquid which has escaped from the blood-vessels accompanied by a quantity of white and red corpuscles which coagulate in these alveoli. Whatever view we take, the blood-vessels are gorged, red corpuscles escape, or break up, and the secretion is rendered red. In later stages of pneumonia, the fibrous walls of the alveoli contain an exudation of cells, and these may, in all likelihood, have had their origin in the fibres themselves.

The stage of purulent infiltration.—According to the view which may be taken of this condition it may be regarded either as a sequel to the forms of inflammation just described or as a true fourth stage of pneumonia; indeed, if the hyperæmic state be regarded as a recognisable condition, the purulent infiltration would constitute a fifth stage. It is better, however, to be content with the three which are usually adopted. With the third stage, or that of grey hepatization, the inflammation has reached its height, and the attendant fever is departing; resolution takes place, the exuded lymph undergoes disintegration, the cells become granular and fatty, and thus the inflammatory products are removed by absorption or expectoration. Sometimes a simple absorption takes place, and the exuded matter may be taken up as rapidly as it was thrown out; this is certain from the frequent absence of expectoration and the return of the diseased lung to its healthy state. More frequently the secretion is thrown up, and the subsidence of the inflammation is shown by the change of the rusty viscid mucus to a thin and yellowish one. It is during this period, when the exudation is softening and passing into the tubes, that pneumonia is usually fatal, and it is frequently observed that the sputum, instead of becoming lighter in colour, has become purple or black. When a post-mortem examination is made and the lung said to be in a state of purulent infiltration, it is found not to have lost its solidity, for it will still sink in water, but when cut through, instead of being dry, it exudes a dirty coloured fluid of a greenish-brown colour mixed with air. It is the hepatized lung softening and saturated with the fluid products of the exudation matter which has broken up. Considering that in fatal cases of pneumonia this is the condition generally found, the stage of purulent infiltration has been regarded by some as a necessarily mortal one; that instead of a simple disintegration of the cellular exudation having taken place, a further infiltration into the alveolar walls has occurred, together with a loss of elasticity and softening which has precluded the possibility of recovery. Yet there are those who, with Hughes Bennett, regard a change resembling this as a necessary termination of an inflammatory process, and, therefore, that a purulent stage of pneumonia is an all but essential one. Of course they would not imply, nor is it implied, that in the fatal form the term purulent is equivalent to the word suppurating; it signifies merely that the inflammatory cell or leucocyte becomes granular and fatty, and, as some would further say, a pus-cell, but it does not mean that anything like

purulent matter is seen in bulk or that abscesses are ever formed. Suppuration, in the ordinary sense of the word, does not take place as one of the stages of inflammation of the lungs. If an abscess is found in the lung the inflammation has not been of the ordinary or croupous variety, but it has been lobular and pyæmic.

It is a point of great interest to ascertain when the recoverable stage of pneumonia has passed and the fatal one begun. It may be true that the disintegration of the new products is necessary, and thus theoretically a purulent stage must exist, but, as a matter of fact, numerous râles throughout the chest are regarded as unfavorable during the recovery from pneumonia, whilst the most favorable cases are those where absorption quickly takes place without any moist sounds. These are the reasons against thinking that the purulent infiltration usually found in fatal pneumonia is a stage which is reached in cases which recover; the probabilities are in favour of the hepatization in most cases not even progressing far beyond the red stage.

Symptoms.—Pneumonia commences generally with the symptoms of a common cold, but which rapidly develop into those of a high fever; there is shivering in the first instance followed by a quick throbbing pulse, furred tongue, hot skin, loss of appetite, headache, delirium, sometimes pain in the side and considerable oppression of breathing. The face is flushed, eyes bright, a cough soon to be followed by a characteristic expectoration of a rusty colour. At this time the fine crepitation is to be heard on applying the ear to the chest. After a few hours this makes away for tubular respiration and bronchophony, whilst the chest is dull on percussion.

I will now speak of these symptoms more in detail. First, you will observe that the fever sets in with great violence, and if there be sickness, shivering and pains in the limbs you may suspect the onset of pneumonia. The *shivering* is often very marked, more so than in any other disorder, excepting ague and suppurative processes. The *tongue* almost from the beginning is covered with a thick creamy fur; the pulse is quick and strong, 100-120. The skin intensely hot, so that the term *pungent heat* has been applied to it. It was formerly supposed that the temperature was higher in pneumonia than in any other febrile disorder, but since the thermometer has come into use it has been found not to exceed, as a rule, 104°. The sensation, therefore, given to the hand of excessive heat must be due to the dryness of the skin or other causes. I said, the face is flushed and the eyes bright, and that the

physiognomy of the patient is important to note; it is sufficiently marked for you to say at once that the case is one of pneumonia, for whereas in typhoid the patient would look dull and heavy, with a lack-lustre eye, the patient with pneumonia would have a flush on the cheeks, and the eyes would be injected, bright and prominent. It is not unusual, also, to see an herpetic eruption on the upper lip. Having now noted the expression, observed the high state of fever and the oppression of breathing, you examine the chest.

The lower part of the lung on one side, is the most usual place where you would hear fine *crepitation;* this is the fine continuous sound like the rubbing of a lock of hair between the fingers, which I have already described. You will remark that the inflammation rarely affects a whole lung simultaneously, but that one portion is first attacked, and then the pneumonic process runs through the lung from base to apex, or apex to base. You next note the character of the *expectoration*, remembering it is by no means an uncommon circumstance for expectoration to be altogether wanting. When present the sputum is rusty and viscid. You will observe the physician, in going round the wards, taking up the spitting pot, and inverting it in order to see if the sputum will run out; if it does not fall out it is the viscid expectoration of pneumonia; a homogeneous secretion always comes from the alveoli of the lungs, and thus contrasts with the frothy mucus of the bronchial tubes. The secretion is also rusty; this means that it is intimately mixed up with the colouring matter of the blood; and is another proof that the inflammatory product has emanated from the intimate structure of the organ. In inflammation you know there is stasis of blood in the vessels, and very often blood escapes and mixes with the secretion; that is, some red globules have accompanied their fellow white ones in wandering through the walls. If there is no actual blood some disintegration of the blood corpuscles has taken place, and the hæmatine becomes incorporated with the secretion. You see the same thing in the smoky urine of nephritis. This rusty expectoration and fine crepitation may be regarded as diagnostic, or, as we say, pathognomonic of pneumonia. I said there was sometimes pain in the side. I am inclined to follow the teaching of Addison in this matter, and say that in simple pneumonia there is no actual pain, but if present it indicates the co-existence of pleurisy; I do not allude to that very slight affection of the pleura covering the inflamed lung, which is almost always present, but a distinct costal pleurisy. There can be no doubt that if there be very acute

pain in the chest pleurisy must certainly be present, and it is equally certain that pneumonia constantly occurs without pain, therefore in all probability the proposition is true—that pain of any severity means that pleurisy complicates pneumonia.

After a short time, that is, in a day or two, you will find the physical signs in the chest are much altered, the crepitation has disappeared, and no sound is heard at all in the tissue of the lung, but only the air passing in and out of the bronchial tubes, and this we call bronchial or *tubular breathing;* at the same time, if the patient speak, the voice comes direct to the ear, and we have *bronchophony;* on percussion the chest is dull, with a feeling of resistance, or sometimes the note is woodeny and high-pitched, and, therefore, occasionally students are heard asking whether the sound should be called dull or hyper-resonant. When the patient speaks, the hand should be placed on the chest, when the *vocal fremitus* is very usually found to be increased in intensity. The *respirations* are of course quickened, being often thirty to forty per minute. Occasionally *jaundice* accompanies pneumonia, and this occurs when the pneumonia is on the left side as well as on the right, and, therefore, the explanation of its occurrence by the transmission of the inflammatory process through the diaphragm, or by pressure on the vena cava, will not hold good. The *urine* is remarkable in being deficient in chlorides; whether this is owing to the salt being all used up in the lung or whether this absence occurs in pneumonia in common with some other febrile complaints, has not yet been determined. We test the urine by adding a little nitric acid and then nitrate of silver. Sometimes albumen is found.

I have said, if inflammation attacked the whole lung simultaneously, it is possible that the duration of the process would not exceed forty-eight hours, but since the inflammation begins at one spot and travels through the lung, the whole duration of the inflammation is generally several days. After the fine crepitation has passed into the tubular respiration, denoting hepatization of the lung, rapid recovery takes place, the dulness on percussion becomes diminished, the tubular breathing less, until the normal resonance and breath-sounds are again heard. In some cases, if the patient be examined exactly at the right moment, a crepitation may again be heard as the disease undergoes resolution; this is called the *redux crepitation.* It is not often heard, because at the same time much of the effused secretion is poured out into the tubes, and bronchial râles then occur, which quite obscure the finer

sounds of minute crepitation. If these moist sounds, however, are heard in the bronchial tubes, they are not very extensive if the patient recover—universal bronchial râles being regarded as a bad symptom. It is not at all uncommon for resolution to take place without the occurrence of any moist sounds. The lung indeed has become rapidly solid, and then as rapidly recovered itself, without the process being apparent by the discovery of any form of crepitation, at the same time there may have been no expectoration whatever. If secretion has been present, and the case terminate favorably, it is seen to undergo a change after the inflammation has reached its height, the sputum becomes less rusty by changing to lemon colour and then to white; it at the same time becomes more watery, until at last it resembles gum-water. Whilst these physical signs are altering, the febrile symptoms are departing as denoted by the fall of temperature, lowered pulse, cleaning of tongue, &c.

If the case should take an unfavorable course, we find, after the disease has reached its height, and the lung has become hepatized, whilst the fever symptoms have abated, and we are regarding it hopefully, that the patient again loses his strength, and that the expectoration continues, together with râles in the chest. Now, in the favorable case the expectoration becomes less coloured until it is white, but in the unfavorable one we observe that the sputum becomes more dark, from rusty it turns to dark purplish black or prune-juice colour, and then to that of liquorice juice or absolute blackness. Expectoration of this colour is the very worst omen we can have in a case of pneumonia; at the same time the simple tubular breathing has changed into bronchial râles throughout the chest; in a day or two more, the patient dies and his lungs are found in the state of purulent infiltration already described.

Let me draw your attention to the importance of noting the general febrile symptoms in pneumonia, as well as the physical signs, and let me add that the same remarks are equally applicable to other diseases. You may, for example, be called to a patient in whom all the physical signs of pneumonic consolidation are present, but you want to know whether the disease is still progressing, or has reached its height. Now this can only be told by observing the presence or absence of febrile symptoms denoted more especially by the temperature. If this be normal you know that the inflammation has ceased, and no active measures are needed. The same remarks, I say, apply to other diseases, as, for example, phthisis. The amount of actual disease in the lung, such as a

cavity, is not so diagnostic of the patient's real condition, as the presence of febrile symptoms, which indicate the progressive nature of the malady. A phthisical patient, with febrile disturbance, without much physical evidence of disease is in far worse condition, than another who has indications of a large cavity, and no constitutional disturbance. Therefore, do not forget to note both symptoms and physical signs in all cases, and not devote all your attention to the latter.

Diagnosis.—It is scarcely worth while to contrast pneumonia with other diseases, for, if you know the symptoms attaching severally to them all, you will not fail to diagnose it. I have constantly seen it mistaken for fever, and I have seen it called inflammation of the brain on account of the delirium, but in these instances the chest had not been examined. At the onset of acute chest disease it is sometimes difficult to form an opinion as to its seat, the patient complaining merely of constriction around the body, and the case may therefore be regarded as one of colic. This has been usually the case when some diaphragmatic pleurisy complicates the pneumonia. The distinctions between pneumonia and pleurisy I shall leave until I come to pleurisy.

Prognosis.—This depends so much upon various circumstances that statistics taken from all cases are of little value. Pneumonia often contributes the final stroke in many diseases, as Morbus Brightii, and constantly complicates other maladies. When it occurs as an acute affection, it may be associated with a pleurisy or a bronchitis. If it occurs in its simplicity, it may involve a lobe, a whole lung, or both. The right lung is most usually affected, and more especially the lower lobe. As I before said, the stages are run through in two or three days, but since the inflammation begins at one part and travels through the lung, the duration of the inflammation in any given case is longer than this.

Treatment.—Pneumonia has been the battle ground of therapeutists in all times. The easily defined and acute nature of the disease caused it to be a promising field for the adventurous trials of enthusiastic physicians, whilst the very fleeting character of the complaint made it above all others the most difficult in which the effects of remedies could be discovered, and thus it has happened that the most opposite and conflicting opinions have been thrown about by those who have waged war on the subject. We have had advocates of antiphlogistic treatment opposed by equally strong advocates of stimulating treatment, whilst a third party has stood by

and declared that all the vaunted means were useless. There was a time when bleeding was always had recourse to and repeated several times, accompanied by medicines consisting of salines, antimony, mercury, and opium. A modified plan of this kind has continued to be the one generally in use until recent times. A few years ago a fashion came into use to treat every one with alcohol, and consequently the only physic for pneumonia was brandy ; of late, Professor Hughes Bennett, by studying pneumonia in a more scientific spirit, has come to the conclusion that this disease, like the fevers of which I have been speaking, runs its course and comes naturally to its end in a few days without any interference on the part of the medical man. If this be found to be true by further observation, I shall certainly enforce it upon you, for all we want is to know the truth of the matter. We are bound to do the best for our patients, irrespective of any traditional sentiments about the necessity of physic giving. I do trust that as educated men you will have so strong a faith in the progress of scientific discovery and truth, that you will always be ready, however old you may grow, to believe that advances are being made in our therapeutic art, and that you will not fall into the category of benighted old gentlemen, who when they see patients recover under a very different treatment than that which they adopted whilst in practice, maintain either that the whole race of men has suddenly changed within thirty or forty years, or that the science and art of medicine has retrograded, and the world is ignorant compared with the enlightenment of the age when they were boys. One is forced to make these remarks when one reads in the journals a letter by a highly respected, though aged physician, declaring that medicine has been retrograding during the last twenty years since he left practice.

Now, however, comes the practical question as to what I should advise you to do in a simple case of acute pneumonia. First of all, don't give brandy as a remedy, except under special circumstances ; as regards the old antiphlogistic treatment, I should like to say as regards bleeding, that there cannot be a doubt from the truthful character of the records, that its use was often attended with a striking good result. We read of persons sitting up in bed with great oppression of breathing, their faces gorged with blood, and the surgeon coming in with his lancet, bleeding in a full stream, and immediately quieting the respiratory process, and relieving the circulation. In such cases there may have been bron-

chitis or there may have been heart disease, but the results were nevertheless the same. This seems to show that in cases of great engorgement of the lungs or of the right side of the heart, bleeding will afford relief and, therefore, under similar circumstances, I should advise you to have recourse to the measure. When you find the venous system gorged in primary affections of the lungs, or in secondary congestions, as in heart disease or from paralytic conditions as in apoplexy, you cannot be wrong in bleeding; you relieve the venous system as well as the right heart, and allow the circulating apparatus to right itself. I have bled patients a sufficient number of times under these circumstances to be able to advise the method with confidence. The objection sometimes made, that the patient is too weak, as indicated by the pulse, is futile, since, owing to the small amount of blood which reaches the left ventricle to be propelled onwards, the pulse is naturally small. Therefore, when I say bleed in pneumonia, it is when the lung is much oppressed and the venous system gorged. I cannot recommend venesection for the reasons our forefathers did, to knock down the disease at its onset, for I have no experience of the treatment under these conditions.

Now, as regards the other parts of the antiphlogistic treatment I feel confident that opium deserves the title; when one sees inflammatory processes subside under its use, I cannot doubt of its influence over them; opium is, therefore, a good remedy, but must be administered with caution, since it acts directly on the respiratory centre to lower its activity. If, therefore, there is not much power of breathing and much mucous in the tubes, it must be given with the utmost care; provided it has no injurious effect in this way, its use is for good. Now as regards antimony, I believe there is sufficient evidence to show that this drug acts also on the vascular system through the nerves, and may, therefore, be called antiphlogistic. As for calomel, I hesitate to put it in the same category; that it has a power of acting on the secreting organs and stirring up the tissues to some kind of activity I have no doubt and thus when various inflammatory results have taken place in the body, mercury is found instrumental in aiding to get rid of them, but whether it has any power of arresting inflammatory processes, I much doubt. Salines are usually given as acting on the skin, kidneys, &c., and as some say for actually cooling the blood. I think there is some evidence to show that the old method of treating pneumonia was followed by as good results as any other, and

having been a witness myself of the method in a great many cases, I have no hesitation in recommending its continuance until it has been satisfactorily proved that all medicines are ineffectual. In a tolerably strong person you may wish to omit the antimony. Under these circumstances you might be content with the saline mixture and Dover's powder. If the patient be feeble, you should give ammonia with the saline and some alcohol, a table spoonful of brandy or whisky with egg every three or four hours. I cannot advise you to give a bottle or two of brandy in the twenty-four hours any more than if I recommend opium or quinine that you should at once administer it indiscriminately and in poisonous doses. I would also recommend you not to use blisters in acute pneumonia, I am strongly impressed with the conviction that they are worse than useless. But you may use the warm jacket by means of hot flannels or spongio-piline tied around the chest. The increased flow of blood to the surface seems to relieve the internal congestions.

I told you that statisticians are showing that the best results have been obtained when no medicine has been given, since the disease must run its natural course. If this be corroborated by further observations, I may at a future time instruct my class to follow merely the expectant plan. There seems no reason, however, theoretically, why the inflammatory process should not be arrested in any one spot, and certainly no reason why it should not be prevented from extending. We meet with cases where the inflammation is limited, showing there are circumstances which affect the process for good or evil. These may be found in the form of drugs and, therefore, I continually use the various substances from time to time recommended. At the present day, veratrium, digitalis, and aconite are undergoing a trial by various medical men, and even the use of cold water.

ATELECTASIS AND COLLAPSE OF THE LUNG

The fœtal lung which has never expanded or contained air, will of course sink in water. A lung also which has once respired will return to its airless or fœtal condition, if death occurs a few days after birth from exhaustion or inanition. Also at a later period of childhood, if the lower bronchial tubes become loaded with mucus, and the child has not strength to eject it, the portions

of lung to which these tubes proceed will become collapsed and airless. In ricketty and otherwise puny children, a little bronchitis will often effectually block a portion of their tubes so that their breathing may become, day by day, shorter and shorter, until they die out with a large portion of their lungs having been for a long time completely idle. In these cases the lung is said to have returned to its *fœtal state*, or to be *collapsed* or to be in a state of *atelectasis* or *apneumatosis*, the last two terms meaning respectively unstretchable and airless. In stronger children who are the subject of bronchitis, portions of lung as individual lobules or groups of lobules become collapsed and airless, and the event occurs in the following manner. If for example, a bronchial tube contains a plug of mucus, this effectually prevents the air entering during inspiration, but readily allows it to escape during expiration. On account of the tube being conical the mucus acts as a valve, and will allow the air to escape past it, though it effectually bars an entrance to its admittance. In consequence of this, the portion of lung to which the tube proceeds is soon pumped dry of air, and is reduced to the airless condition of the fœtal lung. Experiments performed on animals by placing a bullet in a bronchial tube prove how effectually it acts as a valve in pumping the air out of the lung. It consequently happens that in bronchitis a large number of lobules or conglomerations of them may be found quite airless or collapsed. This condition has only of late years been thoroughly understood, although it was pointed out by Bright, in 1828, as met with in the lungs of those who had died of hooping cough. It was regarded, indeed, as indicative of pneumonia, and the airless portions sinking in water were considered to be the subject of hepatization. The difference, however, between these two states may be easily seen, for in the first place if a pipe be placed in the bronchial tube the collapsed lung may be again inflated; and if each portion be examined separately it will be found airless, and sinking in water like hepatized lung, but its cut surface is smooth instead of being granular, and generally wedge-shaped; it is also tough and dry instead of being soft and moist like the hepatized tissue. Moreover, the inflamed part is necessarily distended to the utmost, and if a portion of it were near the surface it would project, and be seen and felt like a distinct hard mass projecting from it; whilst the collapsed part, though red and firm, would be shrunken and sunk lower than the level of the surface of the lung. Sometimes, not only distended lobules but

large portions of lung are collapsed, such as the lower lobe or the posterior part towards its root. I should tell you, however, that although you must clearly distinguish between a collapsed portion of lung and hepatization, yet the two conditions may be associated; for the collapsed lobules may become subsequently the subject of inflammation, and thus you may find a portion of lung which had been primarily collapsed now become partly solidified.

It is remarkable that exactly the same conditions as these are met with in aged persons with this difference, that large tracts of lung become collapsed rather than individual lobules; it may be more especially seen in the portions of the lung towards the root becoming red and quite airless. This may perhaps constitute the "peripneumonia notha" of the older writers.

BRONCHO-PNEUMONIA

This is really a lobular pneumonia and, as I have previously told you, is the disease which is now often styled "catarrhal pneumonia." In the adult the distinction between bronchial tubes and pulmonary tissue is sufficiently great to allow each part to be affected separately by an inflammatory process, but in children (and the same seems to occur in old persons) this distinction is not so marked, and an inflammation may proceed downwards through the tubes to the lung itself. The form therefore of pneumonia met with in children is usually that which is called lobular; the lobar form of adults being more uncommon. The distinction, however, is not merely one involving the amount of lung inflamed, for of late years it has been shown that the pathological or histological process in the two cases is different; that in lobar pneumonia the exudation is more of a fibrinous character, and is derived from the blood, whereas the product of the lobular pneumonia is of a true cellular character derived from a proliferation of the endothelium: a continuation of the process which is taking place in the tubes with which the alveoli of the lung are continuous. Acute lobular or broncho-pneumonia is rarely seen in adults except under special circumstances as in measles; but as a chronic affection, it is less uncommon and frequently lays the foundation of the disease which is known as pneumonic phthisis. Of course I am now speaking of "idiopathic" inflammations, I make no reference to lobular pneumonia as a result of pyæmia. Remember also what I

have already told you, that in consequence of obstruction of the bronchial tubes, portions of lung may become collapsed, and, therefore, in the fœtal inflammations of the lungs of children you may find portions of lung both consolidated and collapsed.

Clinically speaking, this broncho-pneumonia must necessarily be regarded in all its stages, and at first, therefore, it is no more than a bronchitis. A child, for instance, who takes cold on the chest and is in consequence very ill, and said to have an inflammation of the lungs is really suffering in the first place from a bronchitis only; but in very severe cases and probably in nearly all fatal ones the lobules become secondarily affected, and the case then becomes one of lobular or broncho-pneumonia. A post-mortem examination would show the tubes highly injected and covered with a creamy mucus, and if the smaller passages were opened by a fine pair of scissors they might be found completely blocked by it. Sometimes these tubes are temporarily dilated, and the pulmonary tissue between them compressed and airless. A section through the lung may show several portions solidified from hepatization, and other portions firm and airless from simple collapse. In some of the latter, however, inflammatory products may sometimes be found as though an exudation had commenced within them. The collapsed or hepatized parts may vary in size, sometimes occupying large portions of the lung, or being composed merely of an aggregation of a few lobules; occasionally they may be no bigger than a pin's head, and then if the subject of a vesicular pneumonia they may be mistaken for tubercles.

Symptoms.—The child is taken ill with a cold and febrile symptoms, with a temperature of 102° or 103° and very quick pulse, followed by quick breathing and a cough. On examining the chest a loud wheezing is heard all over it both in front and behind, and perhaps a thrill is felt by the hand; if the case be a severe one, the movements are much impeded and a deep depression is seen to occur at every inspiration both at the top of the sternum above and at the epigastrium below. There is a constant hacking cough and some phlegm brought up which the child swallows. In an ordinary case these are all the symptoms observable, but if the child should grow worse it is owing to an extension of the inflammation towards the lobules or to a collapse of the lung. We may generally assume that this has occurred though it may be difficult to diagnose, but if there were any fresh physical signs they would depend upon the amount of the pulmonary tissue involved. Thus on per-

cussion, we might find various degrees of resonance and dulness all over the chest, especially at the back part, and on auscultation râles of various degrees of resonance denoting consolidation beneath.

If the child be delicate and unable to expectorate the mucus, the severe symptoms might be attributed to collapse, rather than to an extension of the inflammation, and more especially should we expect this if they have come on rapidly, if the respiration has become exceedingly quick, and that very little air be found entering the lower part of the lung. The face would in this case be livid from want of aëration of blood; the febrile symptoms might have departed and the extremities would be cold. In these severe cases it is possible that a large part of the lower portions of the lungs may be collapsed, and that besides absence of breath-sound and the presence of a blowing respiration the finger may detect actual dulness over certain parts of the chest on gentle percussion. Children who are the subject of rickets or who have malformed chests are liable to this fatal form of collapse. They may have had more or less wheezing from the time of birth, and then at the age of two or three years speedily die off after a slight attack of cold. On the other hand, the children who have the genuine broncho-pneumonia are often well made with good chests, and in them the severe and fatal symptoms do not come on so suddenly and are attended by more fever.

Treatment.—Now in acute inflammation of the lungs in children, which, as I have explained to you, is really in the first place a bronchitis and subsequently a broncho-pneumonia, I should advise you to adopt an active treatment after the old method, for it is the one which I have found most likely to be successful. This method is to give a saline containing carbonate or nitrate of potash with a few drops of antimony and ipecacuan wine, and at the same time a grain of pulvis ipecac. and perhaps with it a small quantity of calomel three or four times a day; also warmth to the chest and a good warm moist air in the room for the child to breathe. After a time a linseed meal poultice with mustard. If, when you are called in, you find the case advanced and the child very bad, with much difficulty of breathing and oppression of circulation, do not hesitate to apply a leech or two to the chest. I have seen more than one child restored to life by a good bleeding when all hope was gone. If there be much phlegm in the chest you may give an emetic, which not only promotes the evacuation of phlegm from the chest, but the act of vomiting enables the child more effectually to

expand the lungs. There is a difficulty in the use of opium since it tends to lower the respiratory process and prevent expectoration; at the same time it has a great power in subduing inflammatory processes and in sustaining life, and, therefore, is useful when administered with caution. It checks also a harassing hacking cough. You may give a drop or two of laudanum according to age, or a few drops of Tinct. Camphoræ co., or, what is often better, a grain or two of Dover's powder. Do not forget the wonderful effect of a good action of the skin and administer a warm bath where the surface is dry and the breathing much oppressed. If in any case the breathing should become suddenly very quick and there are no marked febrile symptoms and you consequently suspect a collapse of parts of the lung, all you can do then is to administer an emetic, apply stimulating liniments to the chest, and give ammonia and brandy internally.

EMPHYSEMA

This is usually described as of two kinds, the interstitial and the vesicular. The former is rare, and occurs from laceration of the lung and extravasation of air into it. The latter is what is generally meant when the term emphysema is used. It implies merely a dilatation of the vesicles and a breaking down of the partitions of the alveoli so that they run together and large spaces are formed in the lung as in the reptilia; the whole aerating surface is, therefore, much diminished. Sometimes the greater part of both lungs undergoes the emphysematous condition, and then we have before us an affection of the lungs of the most formidable character. In other cases portions of the lungs only are altered by this condition. In this partial emphysema the theories for its production are altogether mechanical, but those who assert them believe that they are sufficient for the production of the universal emphysema I have just spoken of. Of this, however, there is considerable doubt.

In this partial emphysema large vesicles or bullæ are noticed at the edge of the lung, and as they are so often associated with bronchitis and chronic cough the theories for their production are purely mechanical, and are styled, respectively, the *inspiratory* and *expiratory* theories. Laennec had long ago showed that bronchitis and emphysema were constant concomitants; he thought that a portion of lung burst asunder from a retention of air, and so gave

rise to emphysema. It has, however, been proved, as I have already informed you, that the blocking of a tube leads to collapse of the lung and not to its rupture, and, therefore, Laennec's opinion must be erroneous. These more recently ascertained facts of previous bronchitis with resulting collapse were taken by Professor Gairdner and made to account for emphysema, on the supposition that other portions of lung gave way and expanded in order to compensate for the diminution of the obstructed part. On this theory the emphysema would occur during forcible inspiration and would take place at parts of the lung other than those which were connected with the obstructed bronchi. This is so; and as the collapsed parts are found in the lower lobes, so the emphysematous parts are at the upper. This theory is called also the *compensatory* theory because it implies that one portion of the lung expands to take the place of an equal portion which has shrunken. In confirmation of this view it may be said, that independently of bronchitis, if the lung has become contracted in any part from chronic disease, the surrounding parts will be found expanded in order to make up the full bulk. This may be constantly witnessed with the puckered apex of a chronic phthisis.

This theory has been admitted as sufficient to account for the facts in many cases, and, therefore, is probably true, but at the same time emphysema was known to occur in those who had bad coughs and who made violent mechanical efforts in lifting or pulling without any evidence of a previous collapse of the lungs. Another view was therefore taken by Sir W. Jenner, who proved that the lungs are not supported equally by the chest walls, but that there is much less pressure upon them towards the front part. It follows, therefore, that if a person coughs very violently, which implies that he is continually closing his glottis and holding his breath, the lungs are distended to bursting, then it is that those portions give way upon which there is least external pressure, which portions constitute the apex and front edge of the lungs. It has also been shown that draught animals who had had no bronchitis, but who, in the effort to pull, were constantly fixing their chests and in order to do so closed their glottis, were very liable to emphysema of the lungs. You may yourselves have observed that during violent coughing the apices of the lungs may often be observed to protrude above the clavicles, and in the case of M. Groux, who had the fissured sternum, the anterior edges of the lungs were seen thrusting themselves forward every time he coughed. Here, then, seemed a good and

efficient cause for the complaint, and as it occurred in the manner mentioned, it has been called the *expiratory* theory. Although you see it is very different from the other, the two may both be nevertheless true, as they are by no means contradictory of each other.

Those who hold these theories would apparently maintain that the causes which are in operation to start the disease into existence are sufficient to account for its propagation through the whole of the lung. There is, however, considerable difficulty in accepting this explanation, especially when there is no evidence of such mechanical causes as we have mentioned having prevailed in cases of universal emphysema. I allude now to cases where during life you see the whole thorax rounded or otherwise changed in form, and after death the lungs found filling the chest, doughy to the touch, and so distended with air that it can with difficulty be expelled by pressure. At the same time a close inspection shows the vesicles very much increased in size as seen by the naked eye; this is evidently from a rupture of the alveoli, and is a condition found to be universal from one end of the lung to the other. In these cases it is difficult to understand how the mechanical agencies named could have produced the result, and, moreover, in some of the worst examples of the disease there has been no previous history of bronchitis. The breathing has become more and more affected until all the signs of emphysema have disclosed themselves. You must remember, because emphysema and bronchitis are met with together, it does not follow that the former has followed the latter, since the contrary may have been the case. An emphysematous condition of lung by contracting and obliterating the capillaries and otherwise leading to engorgement would of necessity lead also to increased secretion from the bronchial membranes, and so to bronchitis. As mechanical courses fail to account for this general emphysema, it has been thought that they must take place from a simple degeneration of the pulmonary tissue, and therefore it has been suggested that fatty or similar changes might be found to have occurred. This view, however, has not been corroborated by observed facts.

This general emphysema of the lung which demands our attention as a clinical condition is the only one which is productive of symptoms. You may observe how the form of the chest is altered, being rounded and deep from before to behind, giving it a barrel-shaped appearance, the shoulders raised owing to the constant and violent efforts to expand the chest, the upper part becoming, therefore, rounded, whilst the lower is narrow and contracted. The chest,

indeed, in some cases, has the smallest possible motion, for the lung will not shrink and the diaphragm cannot rise. If the cartilages are ossified the difficulty may be still more aggravated. On applying the ear, therefore, the respiratory murmur is faint, but in lieu of it there are very frequently rhonchi of various kinds. These are heard at abnormal times; as the respiratory rhythm has become altered, the act of expiration is much longer than that of inspiration, it is consequently then that the sounds occur. The movement of the chest is accomplished by a rather quick inspiration followed by a slower expiration and immediately upon this a fresh inspiration. There is probably no new morbid sound characteristic of emphysema, for I never heard the crackling which Laennec alludes to. The percussion note is a pitch lower than that of the healthy chest and the sound is said to be one of hyper-resonance.

In this form of emphysema which I have been describing the chest is very large and rounded being expanded to the utmost by the distended lungs. The emphysema is therefore sometimes called *hypertrophous* emphysema to distinguish it from the *atrophous* emphysema of old people. You know that one marked evidence of senility is the loss of the upright position and breadth of shoulders whereby the chest becomes narrow and stooping. This is associated with an actual atrophy of the lung, the partitions of the alveoli waste and the cells run together after the manner of emphysema; but as the whole organ is diminished in bulk, the appropriate term senile or atrophic has been given to it.

INFLAMMATION OF THE AIR PASSAGES

The study of diseases of the air passages is best undertaken by dividing the latter into the larynx with the trachea, the larger bronchi and the smaller bronchi.

CATARRH

The commonest disease of the air passages is a *cold* or *catarrh*; this is characterised by the presence of febrile symptoms, as hot skin, quick pulse, loss of appetite, pains in the limbs, headache, lassitude, and as a result of the hyperæmia of the parts affected, a dryness, irritation, and stiffness of the nose and lips, which is soon followed by secretion from nose, mouth, and mucous membranes of the larger tubes. In a few days these symptoms pass off, leaving the patient well. They may vary somewhat in different persons; in one the frontal sinuses are more affected and the case is one of cold in the head or coryza; in another there is sore throat, in another hoarseness, and in another a slight bronchitis. In some a cold is always attended by "herpes labialis." The combination of the feverish cold with pains in the limbs has suggested the term *febris catarrhalis rheumatica*, and there was once a time when the letters F. C. R. were used in this hospital to denote a bad cold. Sometimes the letter G. was introduced before the C., because gastric disturbance usually accompanies the other symptoms. I need not describe to you the domestic treatment for a common cold. Very often a dose of opium will speedily arrest it, as a few drops of laudanum or ten grains of Dover's powder. I cannot too strongly recommend the old-fashioned recipe of a scruple of nitrate of potash, ten grains of Dover's powder, and two grains of camphor to be mixed together and taken at bedtime.

EPIDEMIC CATARRH OR INFLUENZA

Occasionally an epidemic has occurred amongst us to which we have given this name on account of the most prominent symptoms being those of catarrh. There is, however, a specific element reigning at the time as proved by the wide extent of its influence and from the remarkable constitutional disturbance associated with it. The fatality accompanying the epidemic of 1847, which I well remember, was much greater than during the time of cholera. This was caused by the universal spread of the disease, as the comparative mortality of those attacked was not great. Showing how few individuals escaped I remember on coming to Guy's one day that not a single member of the staff was there, as they were all laid up with influenza.

In most persons the symptoms were those of a common cold, but accompanied by a larger amount than usual of depression, both muscular and nervous, and it was this remarkable depression which characterised the malady, and showed that some specific influence was abroad. There seemed to be a blight in the atmosphere which poisoned every one who breathed it; people felt "knocked down," or prostrated, and sometimes before any special or marked symptoms appeared, they were delirious and even were conscious of being light-headed, whilst they were still able to walk about. Subsequently chest symptoms of some kind or other came on, but then the depression was out of all measure disproportionate to the local mischief. Patients would speak of the oppression at their chest, and of a sense of suffocation when they were suffering only from a slight bronchitis. If you want to understand what the influenza was like, you must think of a common cold or slight chest disorder attended by an inordinate amount of oppression and general debility. In fatal cases there were acute inflammations, as pneumonia, pleurisy, or even pericarditis; the latter then appearing without any rheumatic symptoms. Old persons, already the subject of chest troubles, were speedily carried off.

ACUTE BRONCHITIS

If the cold before spoken of be severe, and the inflammatory process extend down the air passages, the patient is said to have

bronchitis. This in the acute form is not so common as you might think for, bronchitis being generally subacute or chronic, or secondary to a chronic disease. If it occur in a young person, and for the first time, it is a most formidable complaint and always lasts during a period of two or three weeks. There are present the usual feverish symptoms, although these do not run so high as in pneumonia; there is great oppression of breathing, with a sense of soreness or burning in the chest, and a constant hacking cough. The patient often is obliged to remain in the sitting posture in order to respire; there is constant wheezing, and at first only a thin secretion, but this is soon followed by a thick muco-purulent matter from the tubes. This is not so viscid as in pneumonia, nor so watery and frothy as in passive chronic bronchitis, but somewhat intermediate, the viscidity increasing as the pulmonary tissue is approached. The patient remains in this condition for a long time, the temperature not very high, 101° or 102°; the tongue moderately coated, and the skin perhaps perspiring. The breathing may at times be very oppressed and accompanied by a slight lividity with the eyes bright and glassy, and the sense of constriction or soreness very great. It is probable that the trachea ceases to be sensible at the bifurcation, and it is at this point that pain is felt. There is never the acute pain or stitch which is observable in pleurisy. A physical examination shows the chest to be resonant on percussion, but râles are heard all over on applying the ear. These are mostly heard during expiration and entirely mask the natural breath sound. If you could see the tubes you would observe the mucous membrane red, and injected, and velvety, and covered with a creamy secretion.

I should advise you to treat this disease also in the old-fashioned method, by giving a potash saline with ipecacuanha, and in some cases antimony. Also small doses of Pil. Hyd. twice or thrice a day. It has been said that mercury is contra-indicated in inflammations of the mucous membrane, but I have seen the best results follow its administration in the acute forms of bronchitis. The warm room, diluent drinks, you will of course remember.

CAPILLARY BRONCHITIS

This is a form of disease met with mostly in old bronchitic subjects during the severe weather. The patient's breath is very short and

he has a slight cough, but there is little expectoration. The reason is that the larger tubes are unaffected, and thus there are no loud râles nor wheezings heard on putting the ear to the chest, and the air is found entering the upper part of the lung; when, however, you listen over the lower lobes you are conscious that the chest is simply lifted, as you hear no respiratory sound, but in its place a small subcrepitant râle. The air is heard passing in and out of the secretion in the capillary tubes, and at the same time scarcely any of this is expectorated. On looking at the chest once more you will see where the source of the trouble lies; the upper part is violently raised, but the lower is almost motionless or even slightly drawn in during respiration, and the intercostal spaces depressed; for the lung within is almost airless from the blocking of the tubes above: this part may give also some amount of dulness on percussion. You cannot sufficiently bear in mind the existence of this very fatal form of disease, for unless carefully observed you might overlook it both during life and after death. An old person, for example, subject to bronchitis might go out in the cold and actually die in the streets, and you might be called upon to make a post-mortem examination and pronounce upon the cause of death. Unless, therefore, you opened up all the smaller tubes carefully, the object of your inquiry would be altogether unsuccessful. It is remarkable for how long a time patients may linger on with this form of capillary bronchitis, the lower portion of the lung becoming almost collapsed, whilst the upper lobes are alone in operation. The patient lies week after week using only this small portion of his lung whilst the surface of his body is quite livid. During this time he is semi-comatose and often partially anæsthetic—symptoms due to want of aëration of the blood. The remedies are stimulating expectorants; amongst these there is none more efficacious on certain occasions than turpentine. Twenty or thirty drops of the oil of turpentine mixed up with white of egg and a little spirit is one of the best expectorants and stimulants.

CHRONIC BRONCHITIS

This is often subacute, and occurs mostly in persons of middle or advanced age who have been the subject of chronic bronchitis or winter cough. It occurs in all degrees of intensity and degree, dependent upon whether the larger or smaller tubes are affected.

The case, therefore, may resemble that first described; or it may be associated with emphysema, and then we have the chest altered in shape as already mentioned. The auscultatory sounds might of course be of all kinds, such as the various mucous râles, as well as the sonorous and sibilant with prolonged expiration. The upper part of the chest might be heaving, whilst the lower part, together with the diaphragm, might be almost motionless. The expectoration would vary according to the character and intensity of the bronchitis, being watery and frothy with black pigment interspersed, in the more chronic affections of the larger tubes; more viscid and scanty when the smaller tubes were implicated. The febrile disturbance would be slight, but the patient probably would be in bed or keeping his room, having orthopnœa or laborious breathing, giving, indeed, all his attention to the inflation of his lungs.

The treatment in the first place is by salines and expectorants or occasionally bloodletting. In more chronic cases the remedies are very numerous; salines and remedies which promote secretion are generally good, and amongst these iodide of potassium is one of the most useful; we give also chloride of ammonium and the gum resins, also senega and opiates according to circumstances. Counter-irritants, as blisters, are eminently useful, whatever theoretic objections have of late been brought against them. Inhalations are also useful, and lately ipecacuanha has been recommended for this purpose. Besides these forms there is *plastic bronchitis* or that form of inflammation where the whole of the air passages become occupied by moulds of fibrine. These may be expectorated or lead to the death of the patient. Whether this disease is a form of diphtheria is at the present time a disputed question.

The term *fœtid bronchitis* is the term applied to that form where the expectoration is of a highly offensive or stinking character. It is said to be owing to the production of butyric acid or a substance allied to it. You must be ready to recognise it, as patients suffering from this disorder may be thought to have gangrene of the lung; in this latter case, however, the expectorated matters are reddish, whilst in bronchitis they are simply the colour of purulent matter.

In mentioning the above various forms of bronchitis I have spoken often of them as arising from cold or vicissitudes of climate, but they may occur as a complication of many disorders. Thus, in Bright's disease it is the rule to have more or less bronchitis; also in heart disease more or less bronchial congestion, with the accompanying secretion, which results from it. It is also associated with

pyæmia and many of the exanthematous fevers. It may also arise from direct irritants of various kinds. The commonest causes, however, are those dependent upon atmospheric peculiarities, since pulmonary diseases are almost unknown in some typical climates.

INFLAMMATION OF THE LARYNX AND TRACHEA

A slight inflammation, as from cold, produces merely a hoarseness or husky cough, but a further degree of inflammation would be styled *acute catarrhal* laryngitis, a disease characterised by stridulous breathing, croupy cough, and symptoms perhaps of suffocation. This would be speedily fatal or very soon recovered from. Another and very fatal form of laryngitis is often called *erysipelatous;* in this case not only is the mucous membrane intensely injected and swollen, but the submucous tissue, the muscles of the larynx, and the cellular tissue in the neck become involved, and if the patient should live long enough, drops of pus may be found in all these textures. Then there is also *œdema* of the glottis, *tubercular* disease, *syphilitic* and a primary affection of the cartilages themselves known as a *perichondritis.*

CROUP

Inflammation of the larynx is most important in children, and in them it is usually styled *croup.* You are probably aware that in consequence of membranous casts of the trachea having been observed to be thrown off in croup, the disease has been regarded as an inflammation of the windpipe, and called therefore "cynanche trachealis," but there really is no such thing as inflammation of the trachea without that of the larynx accompanying it, and more than this, the symptoms of croup are due essentially to an inflammation and a closure of the glottis. Whenever there is partial narrowing of this aperture a stridulous or croupy breathing and cough result. As it is quite impossible to ascertain whether a membrane is present or not, and as the symptoms are due to the cause named, the term croup has really come to mean a laryngitis. It is simply a clinical term as indicative of certain symptoms. Whenever a child is taken ill and in the course of a short time, say a few hours, becomes hoarse or husky with a peculiar brassy cough and stridulous

breathing, we say the child has croup, or literally laryngitis. It is altogether another question whether the inflammation is simply catarrhal or membranous, that is, whether the mucous membrane is simply covered with a muco-purulent, creamy, corpuscular secretion, or whether it is covered with a distinct fibrinous layer. Again, if the latter be present, whether it is the result of a local inflammation or due to a specific cause known as diphtheria. I have already alluded to this subject, and told you how authors at present differ in opinion. There are those who say the mucous membranes may occasionally take on the membranous form of inflammation, and which they style the croupous form in contradiction to the catarrhal form, whilst there are others who maintain that this membranous form has not a spontaneous origin, but is due to an external specific cause and is indeed none other than diphtheria. The differences of opinion arise because on the one hand in a locality or house where diphtheria exists a case may occur of simple membranous laryngitis not to be distinguished from the "old-fashioned" croup, and on the other hand, because this latter form of croup is often totally unattended by the nervous depression and albuminuria which so strongly characterise ordinary diphtheria. I might also add that the larynx irritated by boiling water will often put on the membranous form of inflammation. It is said that the physical and microscopical differences between a simple croupy membrane and a diphtheritic one are very marked; this is not however generally admitted.

Besides the croupy symptoms arising from cold and evidently inflammatory, they may occur simply from spasm of the glottis, and children not infrequently are suddenly seized with a loud crowing respiration, and with increasing difficulty of breathing, until it seems as if every moment would be their last. This appears to be due to a purely nervous cause and occurs mostly in delicate children. You may remember, however, that in cases of inflammatory croup spasmodic attacks of difficulty of breathing constantly occur. There cannot be a doubt that when mothers tell us that their children are liable to croup and that they treat the attacks themselves, the affection is merely a spasmodic affection of the larynx. The subject of croup therefore stands thus:

- Croup.
  - False Croup or Laryngismus stridulus
  - Inflammatory Croup.
    - Catarrhal.
    - Membranous.
      - Simple
      - Diphtheritic.

If we know a child is subject to spasmodic croup, we tell the mother to dash cold water in the face to bring it to, or if the fits continue, to give an emetic. In chronic cases hydrocyanic acid has been strongly recommended.

When croupy symptoms come on in the course of a few hours after cold and attended by pyrexia, we style the case one of inflammatory croup, but cannot tell whether there be a membrane forming or not. We find the child sitting up in its bed or cradle, with very rapid and perhaps noisy breathing; there may, however, be no marked laryngeal symptoms until secretion collects in the air passages, when a paroxysm of most difficult breathing comes on attended by a loud crowing inspiration which may be heard all over the house. When this has passed the breathing may again be comparatively tranquil, but as the disease rapidly progresses the breathing becomes worse and worse with symptoms of impending suffocation. The child gasps for breath, its nostrils are dilated, the head is thrown back, it is very restless, clutching at its throat or persons around for fear of sudden strangulation, and all this time there is a loud crowing noise. There is probably lividity of the face and all the symptoms and horrors of approaching strangulation. The only noises heard in the chest are the laryngeal ones which everywhere pervade it.

When you are called to a case of croup and cannot possibly know its exact nature I should advise you to adopt the old method, and give an emetic of antimony accompanied by constant applications of heat to the throat; for this purpose there is nothing better than a sponge continually wrung out of hot water. After this you may continue the antimony in small doses with ipecacuan wine. You should place the child in a room so that it can breathe warm and moist but pure air. Emetics subsequently are often good, for during the act of vomiting a membrane may be thrown off and the lungs become expanded. If the urine be found albuminous, it shows the diphtheritic nature of the complaint and the case is generally fatal. If all other measures fail and the child be in danger of suffocation the only thing to have recourse to is tracheotomy. The operation, however, in diphtheritic cases is usually fatal, and in most cases according to my experience where recovery has occurred there has been no evidence of the existence of a membrane. So that my strong impression for a long time has been that the majority of cases of croup which recover, either under the use of medicines or tracheotomy, are cases of catarrhal laryngitis.

I will here remind you of a circumstance which I have dwelt upon before,—that many of the symptoms due to closure of the glottis arise, not from swelling of the mucous membrane, but from paralysis of its muscles. When a muscle is inflamed it is paralysed, as witness the distension of the bowels in peritonitis, the difficulty of breathing in diaphragmatic pleurisy, &c., &c. You may have observed that after a croupy attack and where there is no evidence of any secretion or swelling causing an impediment, paroxysms of bad breathing occur; also after tracheotomy when the larynx has apparently quite recovered itself, that it is impossible to remove the canula, as the larynx will not yet actively dilate; and in one remarkable case I have already mentioned to you death occurred from simple closure of the glottis when not a single morbid appearance could be seen upon it after death. You must remember that during the act of breathing the larynx actively opens by the contraction of its muscles, for if it were not so the glottis would close from atmospheric pressure immediately the chest expanded. Now, this does happen when the recurrent nerves are divided or pressed upon, for then the muscles are paralysed and the larynx is converted into a dead or inactive organ. Exactly the same occurs in inflammation, and especially in diphtheritic inflammation of the glottis, for then directly the child takes a more than usually deep inspiration the rima closes and he is in danger of suffocation.

SPASMODIC ASTHMA

This appears to be a purely nervous complaint, and due to a sudden contraction of the bronchial tubes, whereby air is prevented entering the lung; there necessarily follows congestion, great difficulty of breathing, and the usual accompanying symptoms. That the tubes are capable of contraction is evident from their possessing muscular tissue, and from galvanism being able to produce this effect upon them. In the asthmatic this spasmodic contraction can arise under various exciting conditions, showing that the nerves supplying the bronchi can be influenced through various channels; the phenomena, therefore, are in their nature excito-motor or reflex. I believe that a consideration of the disease on all sides shows that this prevalent view of its nature is correct, but I should inform you that some writers do not admit it, and think that asthma is due to some alteration in the elasticity of the lung. Certain persons only seem liable to these attacks of asthma, and the causes differ in different persons. Those who are subject to it often go to bed well, when after an hour or two they wake up with great oppression of breathing, which obliges them to rise, and in a very short time appear almost in danger of suffocation. The patient may be seen sitting up in bed fighting for his breath, and using every possible muscle to enable him to get air into the chest, or perhaps flying to an open window in order to obtain more air in his struggle for life. He expresses fear of suffocation, and the greatest alarm is depicted on his countenance. Each inspiration is made with an intense effort, whilst the breathing as a whole is not quickened; it may indeed be slow. The chest moves visibly up and down, but scarcely expands, and if the ear is placed upon it but very feeble breath sounds are heard. After a time, owing to the great engorgement of the lungs, a secretion breaks forth from the surface of the tubes; some mucus is expectorated and the spasm is unlocked. Very shortly the patient breathes easily again, and he is well.

Some persons seem predisposed to asthma, as it may attack the young and come on periodically; it is constantly associated with bronchitis and very often with emphysema. The causes are numerous. There are those which act directly upon the mucous

membrane of the bronchial tubes, as cold and dust of all kinds, and therefore asthma is constantly associated with and is a part of bronchitis. The latter, however, may be a consequence of the asthma and produced in the manner before mentioned. Direct irritants of various kinds are frequent sources of asthmatic attacks, as for example in the case of hay-asthma, which is said to be produced by the grains of pollen in the air derived from grasses and flowers. The dust from furs or living animals will produce an attack in some persons. Then, again, irritants acting on other parts of the body by reflex action are sufficient to set up a spasm. This is true more especially of the stomach, so that every asthmatic person is aware how an indigestible or heavy meal is apt to affect his breathing. I have myself seen a gentleman obliged to leave the dinner table owing to his breathing becoming so bad that he seemed in danger of suffocation. Persons will tell you that a late meal or supper is sure to induce an attack, and others, having made the discovery that attacks are warded off by abstemious living, have been led to reduce their sustenance to the barest possible allowance by weighing their food, and some have indeed literally starved themselves. There are cases recorded also where irritation of a particular part of the body has been sufficient to induce an attack; it has been known to alternate with gout, and in some the exciting cause has been overwork or some strain on the nervous system. Indeed the occasional causes for an attack are almost without number.

The treatment of an asthmatic attack is by antispasmodic remedies, or those means which relax the contracted tubes. Those which are at hand sometimes suffice, as hot spirit and water, tea or coffee. Some persons at once take an emetic and this readily relieves, but leaves them exhausted. Amongst medicines we give ether, which may relax the spasm. Also belladonna, but this must be given in a large and almost poisonous dose to have the desired effect; in less urgent cases it is one of our most valuable remedies in small and repeated doses. Inhalation of chloroform will sometimes act very speedily, but it may leave subsequent ill effects. Stramonium still keeps up its character as one of the best antispasmodics when smoked as a cigar or the leaves and stalks in a pipe. Tobacco is very effectual in those not used to it, as it very soon produces nausea and with this a resolution of the spasm. Arsenic has been strongly recommended. I have not used it, but some patients have assured me of its great efficacy when smoked in a cigarette. Nitre paper and numerous other remedies are also useful.

PLEURISY

I have already said that a slight pleurisy accompanies pneumonia in all its forms, whether the latter be lobar, lobular, or pyæmic; it may also be associated with apoplexy of the lung and other local diseases of the organ, or it may be a complication of various constitutional affections, as Bright's disease. It is very probable that, as in the brain, there is an arachnitis pure and simple, and another arachnitis found in association with inflammation of the brain proper, so there is a pleurisy commencing on the costal surface of a simple nature, and another associated with a pneumonia and signifying a deep constitutional origin. In pleurisy, as in other inflammations, the products may consist of lymph, serum, or pus.

In an ordinary case of acute pleurisy a very short time elapses before lymph is poured out, and this is found like a layer of butter spread over a portion of the lung and the surface of the chest; or the exudation may be purely liquid or serum contained in delicate fibrinous meshes. If the process continues, still more fluid is exuded, until the chest is full, and the product may now consist of various proportions of serum, lymph, or pus, or be wholly serous or purulent. If this occurs, then, besides the symptoms arising from the acute inflammatory process, there are the additional ones due to the presence of the fluid and its compression of the lung; if the fluid has been poured out slowly and insidiously, then the symptoms are entirely of the latter kind. It follows, therefore, that under the name acute and chronic pleurisy we have two very different diseases to deal with. The one is an acute inflammatory affection accompanied by considerable constitutional disturbance, the other a chronic disease where general symptoms are absent and local ones alone are present. We will take each case separately.

Acute pleurisy.—The patient generally after exposure to cold or some violent exertion is seized with febrile symptoms and pain in the side. The intensity of this *pain* depends very much upon the

site of the inflammation as corresponding to the degree of the mobility of the chest; for this reason a pleurisy at the upper part corresponding to the upper lobe, may be attended with little pain, whilst pleurisy at the lower part of the chest near the attachment of the diaphragm is attended with the most intense suffering. It will be found on examining the patient that the part of the chest corresponding to the site of the pain is less moveable than the other. This is due to a reflex action; we have no power of our own over the chest to prevent the painful side moving, but the result occurs by an involuntary reflex act; by watching the process of respiration you can see which is the affected side by its less mobility compared with the other. I have observed this in a patient lying insensible. If the diaphragm itself be involved, then there is still less mobility, as if every effort were being made to keep this muscle motionless. This is very important to notice, for the case may be one simply of diaphragmatic pleurisy, affecting either one or both sides, and where no auscultatory signs may exist. Here we must be guided entirely by the quick breathing and the immobility of the lower ribs. Another symptom very frequently present in pleurisy is a short dry cough. In all cases where the pneumogastric nerve is irritated, a cough may occur; it is sometimes one of the most troublesome symptoms and very difficult to treat. The *decubitus* or mode of lying is not of much importance to notice, as it may vary according to circumstances. I have seen patients with so much sensibility of the skin over the inflamed side that they could not bear the bed-clothes to touch it, whilst in others, owing to the acute pain occasioned by the respiratory movements, they have placed a pillow under the affected side and pressed heavily upon it. On *auscultation* at the commencement of pleurisy, a to-and-fro *rub* may be distinguished, and if a thin layer of lymph intervene between the pleural surfaces, *œgophony* is heard when the patient speaks. If lymph be in any considerable quantity, the chest at that part is also *dull* on percussion. The general febrile symptoms are not so acute or severe as in pneumonia, the tongue not noticeably furred, nor the temperature so high. In a simple case these symptoms disappear in a few days and at the same time the pain, dulness on percussion, the œgophony, and the other signs depart. The lymph is either absorbed, leaving the chest healthy, or remains as a permanent thickening of the pleura, or, what is more common, becomes organized, and binds together the lung and the chest wall.

If recovery does not speedily occur, but the effusion continues, the chest becomes full and the patient then begins to suffer from its presence. This fluid may consist of serum, with more or less coagulable lymph or purulent matter. Not only may the chest be thus occupied by the results of inflammatory effusion, but the effusion may take place so quietly and unattended by so few symptoms, that we cannot say that inflammation has existed at all. Under all circumstances the results are the same, as regards the mechanical effects on the organs in the chest, although the character of the products varies under the different circumstances of their occurrence. If purulent matter be present, there will almost of necessity be considerable general disturbance, but if the secretion be purely serous, all constitutional symptoms may be absolutely wanting and the patient be brought to us with no other distressed feeling than shortness of breath. I say effusion may go on so quietly in the chest that the patient has no symptoms until the lung has become compressed, and then he presents himself complaining only of shortness of breath. This may be, indeed, the only symptoms complained of. He may have a short cough, which he himself has scarcely observed, and when you mention the circumstance to him, he may acquiesce in the statement that he can only lie on one side, the *decubitus* in pleuritic effusion being necessarily on the affected side.

Pleuritic effusion.—Since the physical signs are very much the same in all cases, irrespective of the character of the effused fluids and the mode in which they have been formed, we may advantageously take all cases together, and first speak of the physical signs which inform us of the presence of fluid in the chest. No more important case of disease can attract your earnest attention than this, for the ability to ascertain the presence of fluid in the chest may enable you to save a fellow-creature's life, whilst ignorance of the diagnosis will let you stand by and see him die before your eyes. It is for this reason that questions referring to pleuritic effusion and its removal by operation are amongst the most favourite ones amongst examiners, and yet I am sorry to state that satisfactory answers are by no means common. The reasons for this are that when a student is asked what are the physical signs of pleuritic effusion, he immediately thinks of some of the more striking auscultatory names which he has heard, and then having his mind fixed upon the picture of fluid, he gives random and absurd answers about fluid splashing, and the chest bulging, &c. He is

thinking of some positive signs to prove its existence, whereas the signs are really *negative*, and this is the reason they are not impressed on his memory. In health the chest expands, but when the lung is compressed it does not expand; there is *immobility*. In health a respiratory sound is heard, in pleuritic effusion there is *no breath sound;* in health the chest is resonant, when full of fluid it is *dull;* in health there is tactile vibration when the patient speaks, when the lung is compressed by fluid *tactile vibration is absent.* These three negative signs are in themselves almost sufficient to prove the existence of effusion, (1) absence of breath-sound, (2) dulness on percussion, (3) absence of tactile vibration. To this may be added the immobility of the diseased side. If the fluid should still further increase in quantity, then additional symptoms are present, for not only will the fluid displace the lung, but it will forcibly expand the chest, rendering it much larger than it even is in the most forced inspiration.

Under these circumstances the chest may be larger on the affected side, and sometimes the intercostal spaces are on a plane with the ribs, but do not often bulge out; if a bulging is seen at any spot, it does not often arise from the simple pressure of the fluid within, but from the fluid being of a purulent character, and its making an effort to escape by the setting up of an inflammatory process in the walls of the chest. Then, again, the presence of fluid is made known by its mechanical pressure on the surrounding parts, and more especially on the heart, and thus if the effusion be on the left side, which is its more common side, the heart may be so encroached upon as to be pushed over to the right side, where the apex may be felt beating. The pressure on the heart may be one cause of its increased action, and of the sudden death which I have seen occur in such cases. Then, again, from the diaphragm being pressed upon, the spleen may be forced down on the one side or the liver on the other. This cause for descent of the liver should be remembered, as it has sometimes been ignorantly looked upon as enlargement of the liver. As a consequence of the complete compression of the lung, the other lung is doing all the work, and well-marked supplementary breathing is present. I warn you, therefore, against saying that a splash is heard in effusion of the chest, for it is impossible without the presence of the air, and I would advise you also not to say that the chest is larger and the intercostal spaces bulge, for these signs are only present in exceptional extreme cases, and it is long before these occur that the patient's life is in jeopardy

and you may do something for his relief. Then, again, you often say that you know the presence of fluid by the fact of the lung floating about in it, and thus you have resonance or dulness on the front or back of the chest according to the position of the patient. Now, I would ask you not to rely on any such physical sign, and, indeed, it is impossible to occur in conjunction with bulging of the chest, for long before this could occur, the lung would be compressed into so small a space that it could not possibly float to the front or back of the chest from alteration of position. As a matter of fact, this variation of dulness or resonance by position of the patient is not at all common, and is almost worthless as a sign of pleuritic effusion. The lymph is thick and will not allow the lung to move in the manner supposed, as you may any day prove by examining a patient who has pleurisy at the anterior part of the chest, when you will find it dull on percussion, whilst the posterior part preserves its resonance. It is only in exceptional cases, when serum is perfectly clear and thin, that this alteration from change in position can occur, and rarely indeed in pleurisy; it is rather in cases of passive effusion or hydrothorax, occurring in heart disease, that you meet with it. When the chest is full of fluid the lung is drawn upwards and backwards towards the spine, and over this spot bronchial breathing may often be heard.

Now, the next thing to ascertain, if possible, is the character of the fluid effused, whether it be serous or purulent; because if the latter, it will not be absorbed, but must make its exit, whilst the former can be removed by remedies. Now, a great difficulty lies in determining this question, since there are no physical signs to aid us. We must be guided by probabilities, gained from a consideration of the general symptoms. For example, if a patient have acute pleurisy, and shortly afterwards the evidences of effusion are present, we are bound to believe that the matter effused consists of lymph and serum. So, again, if the effusion have come on insidiously, but the patient is able to walk about and is not ill, it can scarcely be supposed that the fluid can be anything but simple serum. If, however, the patient has been ill for some weeks, and he has become thin, his health impaired, and at the same time has hectic symptoms, there can be little doubt that he is the subject of purulent effusion or empyema. Cases, however, are constantly met with where, until paracentesis is performed, we are quite ignorant of the quality of the fluid. The question is one which it would be advantageous in every case to answer, because the treat-

ment very much depends upon our decision. If we believe purulent matter is present, the rule is to evacuate it, for it is useless to look for absorption, this occurring only as a very rare and exceptional occurrence. If the matter be not removed, it will find a channel for itself, either through the chest walls or through the lungs and bronchial tubes; but we prefer not to allow it to remain, in the chance of its taking either of these courses; for it might make its escape into some other channel with very hazardous results.

Treatment and Cure.—As regards treatment, therefore, if we have made up our minds that pus is present from the continuance and severity of the symptoms, we immediately adopt means for its evacuation. In what other cases you may ask do we adopt similar measures? Why, secondly, we tap the chest when the presence of the fluid is endangering the life of the patient. If you are called in to see a patient and find him gasping for breath, and discover that one lung is compressed by fluid, and the other lung, as well as the heart, encroached upon, you have only one duty to perform, and that is, to tap his chest. You need not wait to inquire about the history and symptoms in order to gain knowledge as to the character of the fluid, but you set about removing it. A third reason for tapping your patient is, that when he has been long under treatment and no impression is made on the fluid, you then remove it by instrumental means. Here are, then, three cases or three different occasions in which the profession is agreed upon the necessity for operative interference,—the urgency of the symptoms, the probable purulent character of the effusion, and the not yielding to simpler means. The only case which remains for discussion is that of the recent and moderately acute pleurisy; now here, all are not agreed as to the advisability of paracentesis. It is somewhat a question of fashion, and comes in and goes out like other fashions. When I first knew Guy's Hospital paracentesis thoracis was coming into vogue, and a capital paper was written on the subject, now to be found in the Guy's Hospital Reports, by Mr Cock and Dr Hughes; after a time it came into disuse, it was then again revived; a second time came into abeyance, and is now again being very commonly practised; a further reason, however, for this at the present time is to be found in the improved method of operation and the invention of the exhausting syringe.

If tapping is not had recourse to, we adopt the measures which have always been in use, and by so doing you may observe in many cases that the fluid is absorbed. There is, indeed, a tendency

for absorption to take place if the patient comes into hospital and be kept on low diet. A young man, for example, enters the hospital having had no very severe constitutional symptoms, but his chest is found full of fluid; he is kept quiet and has but little given him to eat and drink, and absorption quickly takes place. As a rule, however, we order him physic, for example, a diuretic saline, perhaps acetate of potash with iodide of potash, and small doses of mercurial. At the same time we apply counter-irritants to the chest, and we prefer the iodine liniment; it blisters the chest, possibly promotes absorption, and at the same time does not interfere with our auscultatory explorations. I believe, myself, the treatment is useful, judging from such a case as we had lately in the hospital; that of a man who had effusion for three months, which entirely disappeared under this plan.

Now, if these measures are unsuccessful we tap the patient; if the fluid be serous and the lung not bound down, it will escape and the lung expand, or we may use a syringe and draw off a certain amount of fluid, leaving the rest to be absorbed. If, however, the fluid be purulent, we often have to adopt a different method; for the lung is coated with lymph, which may prevent the organ from expanding, or from its long compression the tissue itself may have undergone a change; there would be no hope, therefore, of the lung again being restored, and our only object would be to get rid of the purulent matter. If this were universally true, the line of practice would be simple, but there are cases occasionally met with, especially in children, where the exudation has been of a purulent character, and yet the lung expands well on the removal of the fluid by a syringe. In spite of these exceptional cases, I am not sure whether better success would not, upon the whole, be obtained if they were altogether discarded, since the adoption of a method which hopes in every case for a restoration of the lung has been hitherto attended with such unfavourable results. For example, the method at one time was of this kind; to wait until the chest became enormously enlarged and the patient in danger of suffocation, when a trocar was put in the chest and some pints of pus removed; this amount, however, was only the superfluity of the contents of the expanded chest; it had become not only full, but distended, the purulent matter occupying the mediastinum and parts which are not usually filled with lung. It was this superfluous fluid which was removed, relieving the patient from the undue pressure, but still leaving his chest full. After a short period a further

secretion took place, productive of the same symptoms, when the trocar was again had recourse to with a temporary relief for the second time; for a third and fourth time the operation was repeated, when the patient at last died; and on post-mortem examination the chest was found full of pus, and much in the same state as when the patient first entered the hospital. Now, having seen several such cases, it is sufficiently evident that the lung has very little chance of recovering itself in a case of empyema, and yet this is what we are always hoping for. If, then, the lung is irretrievably bound down, it is as impossible for the fluid to escape without letting the air in, as it is for beer to flow from a barrel without the introduction of a vent peg; under these circumstances the practice has been of late years either to evacuate the contents of the chest by letting the air in or to let the matter slowly escape while the chest contracts upon it in the ordinary process of cure. If it could be said that, wherever purulent matter escapes on tapping, it shows a severity and duration of disease which must have irrevocably spoiled the lung, such a practice as I have named might be recommended to be invariably followed, but since occasionally in young persons the lung is capable of recovering itself, we first try the effects of withdrawing the fluid by means of a syringe.

I should advise you first to attempt to draw off the fluid; if the lung does not expand, then you must introduce a drainage tube. This is done in various ways. For the introduction of the trocar you choose a position at the side of the chest, between the fifth and sixth rib, and between the digitations of the serratus; you are told to pass the instrument along the upper side of the rib, so as to avoid puncturing the intercostal artery; I don't think this direction is often followed, as there is often only just space sufficient for the introduction of the trocar; but I have known the artery wounded and attended with much hæmorrhage into the chest. After removing the trocar you may thrust into the hole a piece of india rubber piping, and allow the other end to fall into a vessel of water: the matter will thus gradually run out. Another operation is to make two openings and pass a piece of perforated pipe between them. The first method of allowing the pipe to fall into a vessel of water is now very commonly adopted, for if the lung is capable of expanding, the dilatation gradually goes on as the pus drains out, and a perfect recovery may take place; but if the lung should be incapable of expansion, then as the matter escapes, the chest falls in and a cure is effected at the expense of a damaged lung.

The same process occurs when the matter is self-evacuated by making its way through the chest-walls; a cure can only be completed by the chest-wall falling in to grow to the lung, and the process is naturally a very long one. In the case of an abscess in the soft parts, the surgeon can compress its sides after the evacuation of the matter, but an abscess in the chest having bony and unyielding walls, will go on secreting matter into the hollow which has been emptied, and the process will continue for many months until the cavity has completely contracted. This method of letting the matter run through a tube or a fistulous opening in the chest is not always sufficient to relieve the patient, and I have now seen several lives saved by the adoption of bolder measures. It happens sometimes that the patient is really suffering from the presence of a large quantity of purulent matter in the body and the drainage tube does not readily enough assist its discharge, we must then open the chest and allow the matter to escape as in the case of any other abscess. A most striking case of this kind was that of a boy lately in the hospital; coming in with empyema, paracentesis was performed, some matter evacuated, and a drainage tube put in the chest; a slow and constant flow took place, but not sufficient to relieve the patient for any length of time, for he sank into a febrile or hectic state, with quick pulse, loss of appetite, and such increasing debility that it was evident his days were numbered. Under these circumstances I proposed to let the matter out by a free opening, and I called in consultation my colleague, Mr Durham, who seeing the dying state of our patient, thereupon made a long incision in the chest; immediately the air rushed in and the matter gushed out; the chest was further cleansed by a quantity of carbolised water being pumped in; and this was repeated every day. The improvement was instant and most remarkable; in three days the temperature was reduced to normal, and the appetite had returned. In a few more days the lad had left his bed, and very soon was walking about the grounds. It was interesting for the students to examine his chest; for of course when percussed it sounded like a drum. After being in the hospital some time, and it being found that the chest was slowly contracting, he was allowed to leave and perfect the cure himself, he having been taught to wash out his own chest; he would place a funnel in the fistulous opening and pour water into it, and then turning on his side let it again run out, until the cavity was cleansed. About the same time we opened a woman's chest where the drainage tube was ineffectual in

relieving the constitutional disturbance. In her case, as well as in the young man's, I have no doubt that her life was saved by the operation.

You will see, then, what are our operative procedures. 1. In all cases where life is threatened you perform paracentesis. 2. In all cases of long standing effusion where ordinary measures have failed. 3. In all cases where you suspect purulent matter. Whether you should do it in more recent cases, where the symptoms are not urgent, is the only question still open in the profession. Since we have had a good exhausting syringe, I have certainly done it more frequently than heretofore, but I cannot recommend its use indiscriminately in all cases, seeing that rapid recovery may often take place without any operative measures whatever.

If purulent matter be present in the chest, and you do not evacuate it, it will spontaneously burst its bounds and escape; it may take a course injurious to life, as into the pericardium or into the abdomen, but most usually it makes its way through the chest externally or through the lungs internally, the latter is the mode which is followed by the best cure. If it passes through the chest-wall it excites inflammation in the parietes, an abscess forms, which opens, and so allows the matter to escape. It is worthy of remark that although we may tap the patient low down for the sake of safety, yet that Nature prefers the upper anterior part; thus you will see the natural fistulous opening more commonly in the front and upper part of chest. As cure takes place the chest becomes contracted and sometimes the spine is bent over on that side, whilst the other side of the chest becomes enlarged owing to the hypertrophy of the lung.

If the matter be evacuated through the lung, there may be no previous warning of it, but the patient suddenly coughs and ejects some pints of matter. This may continue for some days or weeks and complete recovery ensue. If a large tube be opened it may happen that air escapes into the chest, and then we have a *pleuro-pneumothorax* to deal with; known by the physical signs before mentioned, the metallic sounds on coughing and the splash on succussion. It is a remarkable circumstance that sometimes on post-mortem examination of cases, where evacuation of matter has taken place through the lung, no opening can be discovered, and this suggested to my late colleague, Dr Barlow, the possibility of a rapid secretion taking place from the bronchial membrane, of matter whose pabulum was supplied by the pus on the outside of the lung. The fact has often

been observed that after paracentesis and the pressure removed from the lung, a bronchial secretion will immediately occur, showing some connection probably between the stagnation of blood in the lung and the secretion from the bronchial membrane. You may remember that the simplest, safest, and most effectual recovery occurs through the natural evacuation of pus through the lung.

PNEUMO-THORAX

Pneumo-thorax, medically speaking, is the case where air escapes into the chest from the lung. This implies of course rupture of the lung from softening of the tissue, and may arise either from disease commencing within the organ or from without in connection with a pleuritis. The former is the more common cause owing to the giving way of a small abscess or softening tubercle in early phthisis. The patient may have had previously pulmonary symptoms or not, when he is suddenly seized with difficulty of breathing and sense of suffocation, or in other cases which are perhaps equally common the air is gradually pumped out of the lung and the sense of dyspnœa comes on gradually. These cases are often the worst, for, instead of the chest being simply filled with air, it becomes gradually and tightly filled to the utmost state of distension.

The cause of the symptoms mentioned is sufficiently evident on examination of the chest, when one side is found tympanitic on percussion, with a total absence of respiratory sound on applying the ear, as well as no tactile fremitus. There may be heard instead, a blowing or amphoric sound, as of air entering a large space, but this only occurs occasionally, and therefore the characteristic sounds are not brought out unless the patient coughs. Then a most remarkable hollow and almost metallic note is heard clearly denoting the existence of a large cavern of air within. If the opening from the lung is closed this metallic sound may be wanting, and then we have to produce it by another mode of our own contrivance. By placing a coin on the chest and striking it, a deep bell sound is produced; this is called the "bruit d'airain." Occasionally the air is gradually pumped out into the chest until it becomes tightly distended; the chest bulges and is round; the patient lies on that side; and if the pneumo-thorax be on the left side the heart is pushed over to the right. You have, indeed, many of the same physical conditions as when fluid is present.

In these cases of pneumo-thorax the air may be absorbed and the patient recover, but it often happens that a pleurisy is set up in consequence of the rupture, and then we have the case of pleuro-pneumo-thorax. The physical signs of this condition are well marked; they are those of the presence of air in the upper part of the chest before mentioned, and also those of fluid below, the division of the two being made distinct by the resonance above and dulness below. Also when the patient coughs some fluid may be displaced, and we have the sign known as "metallic tinkling." There is also the splash to be heard on succussion, or when the patient is shaken.

The other cause of pneumo-thorax is where fluid has burst from the chest into the lung, and air has escaped back again into the chest. Here we have a pleuro-pneumo-thorax from the beginning. In this case, as before, there may be the splash on succussion, the metallic tinkling, and amphoric resonance; but on percussion it is not usual to get a resonant note over the part containing air, for the reason that the costal pleura is thickly coated with lymph, and fails, therefore, to give out a clear note when struck. A stronger stroke might produce a tympanitic note, and then you would have a similar dull tympanitic sound as you would have over a cavity in the lung.

In these cases recovery may gradually take place by contraction of the chest. In sudden and urgent distress, where the lung has ruptured and the chest tightly distended with air, you must draw off the gas by an aspirator.

DISEASES OF THE HEART

Normal Action of the Heart. — Before you can understand the method of diagnosing the morbid sounds of the heart, you must recall the physiology and action of the organ. It is necessary that you should carry this knowledge with you into the wards, and therefore I will first refresh your memories on the subject. To understand the action of the heart we may first make the matter more simple by considering that the organ is double, and therefore we may take each half, the right or left side, as a perfect heart in itself. We thus have an auricle, ventricle, and large vessel of exit to deal with. The simplest idea of a circulating fluid is to be gained from the old experimental instrument, where an endless tube, with an elastic bag in one portion of it, is constantly sending the fluid round and round as the bag is compressed, and then if a porous substance be placed within it on the opposite side to this bag, the fluid will pass onwards to it in jerks and return through the porous substance in a continuous flow. If, instead of the porous material we suppose a system of capillaries, we gain some idea of the circulating process, and if further we make a dilatation of the end of the tube before it enters the bag (which corresponds to the ventricle), in order to collect a sufficient amount of fluid to send onward again at a fresh stroke, we have produced an auricle. In an apparatus of this kind, you see the fluid pouring into this dilatation or auricle, and at the same time into the elastic bag or ventricle and so on to the vessel or aorta. The auricle is nothing more than the dilatation of the vessel which carries the blood to the ventricle so as to completely fill the latter before it contracts; it is surrounded by muscular fibre, and it has a separate rhythmical movement of its own.

The action is this: the blood is pouring into the auricle and

through its opening below into the ventricle until both these cavities are nearly full of blood; then the auricle quickly and momentarily contracts, so as to drive more blood into the ventricle, which then takes on the action and propels it into the aorta, which, again reacting on the blood, drives it still further onward. The order of succession of the movements appears to be self-evident, and can scarcely be otherwise than I have described. Difficulties had been made by looking at the heart as a complex organ, but regarding it in this simpler light there can be but one order of motion. If a contractile tube is propelling its contents, the movement must be regular and onward. In the intestine the peristaltic action is of this kind, and in the case of the stomach we believe contraction takes place regularly from end to end, in repeated circles; so also in the uterus the contractions begin above and pass down in zones. In a simple blood vessel the blood would be propelled onwards in this rhythmical way and it would in nowise be altered because a part of the vessel was occupied by a large muscular contractile bag called the ventricle of the heart; nor would its action be altered by the tube being dilated at its end into a sac called the auricle. The fluid would first enter the auricle, then the ventricle, and then the aorta in regular succession. The blood is, in fact, pouring down into the auricle and through the auriculo-ventricular orifice into the ventricle, the mitral valve floating up in the fluid, when the auricle suddenly contracts, driving more blood onwards into the ventricle, which then itself contracts more slowly, propelling the blood into the aorta, which again contracting on its contents sends it still further onward into the system.

That this is the action of the heart, is proved by the cardiograph, an instrument which may be placed over different parts of the heart, and the tracing of their movements indicated. The index over the auricle makes a sudden rise and as quick a fall, the index over the ventricle does not move until that of the auricle has nearly fallen and after this comes the index of the aorta; the series then again begins, but remember that the auricle does not move until the aorta has ceased to contract; therefore, the contractions of the auricle and aorta are not synchronous. If an index be placed over the ventricle alone, the order of movements can also be seen, for although it is only moved violently during the contraction of the ventricle, it is slightly jerked upwards previously by the contraction of the auricle and it receives also a slight jar afterwards

by the contraction of the aorta. The rhythm of the heart's action can thus be easily demonstrated. If the action of the heart also be represented by a circle divided into fifths, we should find that nearly two-fifths are occupied by the contraction of the ventricle, nearly one-fifth by the contraction of the auricle, and nearly the remaining two-fifths by the refilling of the auricle and ventricle. The short time which is left, deducted from the periods of fifths, is occupied by the momentary contraction of the auricle. If you watch the movements of the heart in a living animal, you will observe a deliberate movement of the ventricle, followed by that of the aorta, but the auricle so momentarily contracts before the ventricle that it looks as if the movement of the one passed into that of the other in a kind of screw-like motion. The whole action of the heart indeed is screw-like ; the ventricle is spiral, and as it contracts, the blood is sent spirally into the aorta which then takes on the same action, and the sigmoid valves close down, not simultaneously, but one after the other, fitting together with a screw-like motion. The four large valves or orifices of the heart are arranged one around the other, in a similar spiral manner. This spiral movement of the ventricle is due to the arrangement of six or seven distinct layers of muscular fibres, crossing one another. The use of the papillary muscles is clearly for keeping the valves tight during the contraction of the ventricle.

The contraction of a cavity of the heart is called its systole, and its opening or expansion the diastole. Now, if you place your ear over the heart you will hear two sounds, one dull and prolonged and the other short and sharp ; the first corresponds to the contraction of the ventricle and is called systolic ; the second to the contraction of the aorta, which happening during the opening of the ventricle, is called diastolic. For you will observe that we cannot unfortunately call the sounds after the conditions which cause them, for such has not been the practice, but we call them after the state of the ventricle at the time of their occurrence ; thus, this second sound is not named after the condition of the aorta by whose contraction it is caused, but diastolic, after the ventricle, and in the same way the sound produced by the contraction of the auricle, in some forms of disease is not called auricular, but presystolic, because happening before the systole of the ventricle. This nomenclature is unfortunate, for it not only prevents us from naming a sound after its true cause, but is wrong in leading us to think that the names we use accurately correspond to the state of the ven-

tricle, which they do not. I told you that after the ventricle contracts, it again opens, and remains open while it is being filled with blood, and during this period the aorta is contracting and the auricle is contracting; therefore, whatever occurs in the heart's rhythm, which is not systolic, is diastolic, and yet we have limited the term diastolic to that first portion only of the heart's dilatation, which corresponds to the aortic contraction or second sound of the heart. At the present time the term diastolic is used by most writers to indicate only that period of the heart's action which corresponds to the second sound, although there are others who use it it in a larger sense to denote the whole period of the heart's dilatation.

The first sound of the heart occurs during the contraction of the ventricle, its blow against the chest-walls, the flow of blood into the aorta, and the closure of the mitral valve. Most physiologists believe the stretching of the mitral valve is mainly instrumental in its production, influenced of course by the condition of the muscular walls of the ventricle to which the valve is attached. The sound, therefore, although mainly produced here, is heard best at the apex, because the base of the ventricle lies deep in the chest, whilst the apex is tilted forwards to the chest-wall to which point the sound is carried. The second sound is heard best at the base over the region where it is produced; it has been demonstratively shown to be due to the closure of the sigmoid valves.

You will observe, then, that we have a means of knowing the causes producing abnormal sounds by the period of the heart's rhythm in which they occur; and also from the position of the sound in relation to the heart, all murmurs, for example, produced at the mitral orifice being heard best at the apex. These facts would be true in whatever part of the body the heart might be placed; but as it is always accurately fixed in one spot, we have a further mode of ascertaining the origin of particular sounds, by discovering the place in the chest where they occur and the direction they take. Now, if we place the heart in position, we shall find that if a line be let fall along the left side of the sternum two-thirds of the heart are to the right of this line and one third to the left. The base of the heart is at the third rib, or still higher if we reckon the appendages. The apex is at the upper edge of the sixth rib, and is formed by the left ventricle, it is found about two inches below the nipple and a little to the inside; the nipple being placed very uniformly on the junction of the fourth rib with its cartilage.

The area of dulness is about two and a half inches in each direction over the lower part of heart, the lung covering the upper part. Now, as regards the position of the valves, you must remember that they lie one over the other, which you seem sometimes to forget when you say in your reports that you place your stethoscope over this or that valve, which is impossible, for the stethoscope cannot cover one without partially overlapping another; therefore, the origin of the sounds is not known by the position of the valves, but by another method; it is by the conduction of the sound, in any given direction by the current of the blood. The valves are situated about the third costal cartilage, a little above and a little below. The *pulmonary* valves are at its upper edge; the *aortic* behind them as well as a little below and inwards towards the median line. The *mitral* is at the third intercostal space, and a little to the outside of the aortic. The *tricuspid* is in front of the mitral, and is nearer the median line behind the sternum. The aorta passes to the right, the pulmonary to the left side of the chest. The sounds, therefore, produced at the valves are best heard at the second right costal cartilage; the second left costal cartilage, or a little below; the fifth right costal cartilage, and the apex of the heart respectively. Now, remember the rhythm of the heart before we pass to the morbid sounds.

Filling of heart and contraction of auricle—Period of silence.

Contraction of ventricle—First or systolic sound.

Contraction of aorta—Second or diastolic sound.

Filling of heart and contraction of auricle—Period of silence.

Diagnosis.—Now, we will pass on to the *morbid sounds*. These are mainly due to the changes in the valves, at the auriculo-ventricular orifices and mouths of the large vessels. You can easily understand how, if these valves were impaired, a leakage would occur, and also how they might as a result of inflammatory contraction cause an impediment in the channel of the blood. It would follow from this that at each orifice there might be a condition which should cause obstruction, and an imperfection of the valves which should cause regurgitation, and that there would be no reason why these two conditions should not occur together at the

same orifice. Since, then, there are two hearts, we have eight possible morbid states, equal to the production of as many morbid sounds or bruits. Practically, however, these are almost entirely confined to the left side.

Now, let us suppose the auriculo-ventricular orifice contracted (and which would really be the mitral valve) the blood would meet with a difficulty in its passage through it, and a bruit would be produced; this would necessarily occur during the contraction of the auricle, and would be, therefore, an auricular systolic bruit. It would take place during the normal period of silence, and before the first sound of the heart.

If the same orifice possessed an imperfect valve, regurgitation would occur, and a bruit would be produced, necessarily occurring during the systole of the ventricle and first sound of the heart.

If the larger vessels were obstructed, which would be practically the aorta, a bruit would be produced, which necessarily would also be produced during the ventricular systole.

If regurgitation took place through the same orifice, a bruit would be produced during the time of the contraction of the aorta or second sound, and which happening at the period when the heart is dilating, is called diastolic.

The morbid sounds thus produced are named after the condition of the ventricle at the time they occur, and, therefore, two of them would be systolic, one presystolic, and one diastolic, although, as before said, the presystolic might with justice be called diastolic, and it is so named by some authors.

Obstruction at auriculo-ventricular valve: auricular contraction: presystolic sound: natural period of silence.

Regurgitation at auriculo-ventricular valve: ventricular contraction: systolic sound: natural first sound.

Obstruction at sigmoid valves: ventricular contraction: systolic sound: natural first sound.

Regurgitation at sigmoid valves: aortic contraction: diastolic sound: natural second sound.

Now, if we take each heart separately, these four sounds are distinguished, both by the period of the rhythm in which they occur, and by the direction in which the sounds are conveyed. The direction is mainly due to the course of the current of the blood. Thus,

if we take the left side of the heart, and consider the case of obstruction of the mitral orifice, the sound produced by the blood passing through the contracted orifice would be carried downwards to the apex. In the case of regurgitation through the mitral orifice, I told you that all sounds having their origin at the mitral valve were heard better at the apex than over the valve itself, because the latter was deep-seated and the apex tilted the sound forward to the surface on which it struck, therefore the bruit produced by regurgitation is heard better at the apex than over the valve itself, but at the same time it is carried back towards the auricle with the flow of blood, and is, therefore, heard loudly in the dorsal region. Any bruit caused also by obstruction of the aorta would in like manner be carried from its point of production at the orifice of the vessel along the aorta, and a bruit produced by regurgitation would be carried downwards either towards the apex or down the sternum to the ensiform cartilage.

The same rule holds good if we take the right side of the heart, where disease is more rare. The only bruit which is not very uncommon is that produced by regurgitation through the tricuspid valve; this would be systolic in rhythm, and heard not over the apex, but at the lower part of the sternum and fifth right costal cartilage. If the tricuspid were narrowed, there would be, I suppose, a presystolic bruit heard towards the right side. If the pulmonary artery were contracted, which is sometimes congenitally the case, a systolic bruit would be heard at the base of the heart, conducted to the left side, under the clavicle or second left costal cartilage. If regurgitation through the pulmonary orifice should occur, then a diastolic bruit would be heard conducted downwards towards the right heart.

To revert to the left side, to which disease is almost exclusively confined, you may remember that a systolic bruit heard best at the apex and lost as the stethoscope passes upwards, but traced around the chest, beneath the axilla, and heard louder again at the inferior angle of the scapula, between the space, corresponding to the 6th—9th vertebræ implies *mitral regurgitation.*

A systolic bruit heard at the base and carried along the course of the aorta, being very audible at the second right costal cartilage; seldom heard behind, or, if faintly, opposite third and fourth vertebræ, implies *aortic obstruction.*

A diastolic bruit replacing the second sound heard at the base,

and carried downwards either along the sternum or towards the apex, implies *aortic regurgitation.*

A presystolic bruit heard about the region of the apex and almost confined to this spot, implies *mitral obstruction.*

Other diagnostic features of these valvular defects I will speak of when I have described the changes which the cavities of the heart undergo in these respective diseases. Enlargement of the heart, as a rule, is due to some impediment in the course of the circulation. If a muscular organ have increased work to perform, it becomes hypertrophied, a good example of which is seen in the bladder in the case of stricture of the urethra. If there be contraction of any of the cardiac orifices there results hypertrophy of the cavity behind it, as of the left auricle in narrowing or stenosis of the mitral valve and of the left ventricle in narrowing or stenosis of the aortic orifice. If, again, the cavity behind the obstruction is unable after a time to overcome the difficulty of transmitting the blood, it undergoes a dilatation also; much more is this likely to occur where a valve is inefficient, for then a part of the blood flows back into the cavity, which can never empty itself, and becomes necessarily much enlarged. In obstruction of an orifice the tendency is to hypertrophy of the cavity behind; in regurgitation the tendency is towards dilatation. Other circumstances of course come into play to affect the relative proportion of these two conditions, as, for example, the state of nutrition necessary for the growth of muscle, and therefore in a case where the tissues are badly nourished, the heart would be specially inclined to yield when much pressure is put upon it.

Since the heart is a double organ we are obliged to regard the lungs in this question of enlargement, since they lie physiologically between the right and left hearts. Now, if we take a normal heart with its four cavities and beginning on the right side we can see how changes in its size will occur. Since disease rarely happens to the tricuspid or pulmonary valves we must first go to the lungs themselves; now if any impediment occur in the circulation through these organs, as in bronchitis, we necessarily have hypertrophy of the right side of the heart, first of the ventricle and then of the auricle. Passing to the left side of the heart, if any impediment occur at the mitral orifice, there is, of necessity, great enlargement and hypertrophy of the left auricle; so marked is this that a safe conclusion may be drawn from it as to the existence of obstruction at the orifice below it, and this is an interesting fact to remember in

connection with its slower contraction under these altered circumstances. In this case the blood is thrown back on the lungs, and here an obstruction being caused, the right side of the heart becomes hypertrophied as in the case of bronchitis. When there is simple regurgitation through the mitral orifice, the auricle enlarges, but it is not thickened as in the case of obstruction. In aortic obstruction the ventricle is simply hypertrophied, while if regurgitation accompany it there is dilatation added to hypertrophy, and the ventricle becomes very much enlarged; this necessitates after a time an enlargement of all parts of the heart behind it in the course of the circulation, and the whole organ becomes proportionally increased in size, and we have what is sometimes called the "bovine" heart. Several models in the museum were made to illustrate these changes in the different forms of valvular disease.

Now, all these conditions have to be taken into consideration when attempting a diagnosis of cardiac disease; this being mainly obtained by considering at what time of the heart's rhythm a murmur occurs; by the direction in which the murmur is transmitted, and by the position of the bruit as regards the walls of the chest. I should tell you that although for the sake of simplicity, I have described each of these murmurs as occurring separately, any two of them may take place together, for when we say a valve causes obstruction, we do not imply that its imperfection may not also cause regurgitation. Now, this is very commonly the case as regards the aortic orifice, and therefore, whilst there is an obstruction or onward murmur, there is very frequently a backward or regurgitant murmur heard at the same time, and we have consequently a double aortic murmur, the systolic and diastolic being combined.

Next we will take each of these more usual valvular diseases separately, beginning with the *aortic obstruction*. We have here the case where the valves offer some impediment to the flow of the blood, and produce a murmur; at the same time being perfect enough to close and prevent regurgitation. Under these circumstances we have a systolic murmur heard at the base of the heart and carried across to the sternum to the second right costal cartilage. The natural first sound of the heart heard at the apex is not quite so good as in the healthy heart, and is partly covered by the murmur here faintly heard. The second sound also which is produced by the closure of the sigmoids is indistinct. The heart

is hypertrophied, and the apex beat is observed to be lower than natural. In a simple case with marked obstruction, the pulse is often small, slow, and regular.

In *aortic regurgitation*, there is generally present the obstructive murmur just mentioned, and in addition to this a diastolic murmur replacing the second sound; there is, therefore, a double or see-saw murmur heard over the base of the heart where these two sounds are produced; the second or diastolic sound is carried downwards with the current of the blood, sometimes towards the apex, but more generally straight down the sternum, which is almost in contact with the aorta.* The heart is much enlarged, known by the increased area of dulness on percussion, and by the apex beat being much lower than usual, and to the outside of the nipple, to which spot it would be necessarily carried. The pulse is very characteristic in this disease; having two well-marked features, due to the enlargement of the heart, and the incapacity of the aortic valves. First of all the pulse is regular, large, and full, visible to the eye, and to be felt in the smaller vessels, where it is usually not palpable, and secondly, this large full pulse suddenly collapses under the finger; it is called a receding, collapsing, jerking, or kicking pulse, some have called it water-hammer pulse, after a toy of that name. This remarkable recession is often much intensified by holding up the arm above the head, whilst the patient is in the recumbent position. The throbbing of the vessels is often a painful symptom felt by the patient, and may often be observed in the retinal vessels by the aid of the ophthalmoscope.

Mitral regurgitation is known by a systolic bruit, heard best at the apex, and carried round the chest under the axilla to the angle

* I have always explained this by the fact of the aorta being immediately beneath bone which is so good a conductor of sound. If the ear could be placed immediately upon the heart I have no doubt that the sound would be carried down towards the apex according to the law, but from the position of the heart in the chest the sternum conducts the sound to the almost entire occlusion of the more normal one. Dr. Balthazar Foster has sought very ingeniously to explain the different course of the bruits by the particular valve which is affected. When, he says, the left-hand or mitral sigmoid valve is diseased, the blood is carried down into the ventricle and the bruit transmitted to the apex; whereas, if the right-hand and posterior sigmoids are affected, the blood impinges on the septum and the sound is carried down the sternum. He further says that the first-mentioned case is the less important since the sigmoid affected has no coronary artery, and therefore its diseases would not interfere with the heart's supply of blood during aortic systole.

of the scapula, where it also is distinctly audible. The heart is somewhat enlarged; its rhythm may be altered in various ways, and the pulse very frequently is small and irregular.

Mitral obstruction is characterised by a bruit which some call diastolic, but it is more usually styled presystolic, because it is not synchronous with the diastolic sound of heart. It is caused by the current of blood passing through a strictured mitral orifice, is heard best at the apex, and never at any distant spot from it and rarely behind; it is often harsh and prolonged, and has the name "churning" given to it, but often varies much in character from day to day. This obstructive mitral bruit has been generally overlooked until the present time, but its existence is certain, and the proof depends upon its rhythm, localisation, and after death upon the fact of the extreme hypertrophy of the auricle. The principal reason for its being hitherto disregarded has been suggested by Dr Fagge, who has shown how the first sound of the heart is necessarily altered in this disease of the mitral, and that the natural prolonged tone becomes an insignificant short sound, whilst at the same time the slowly contracting auricle produces a lengthened bruit; in consequence of this, the latter sound was regarded as the systolic, and the short systolic as a diastolic sound. I have no doubt that herein lies the true explanation of our former ignorance. A difficulty also against the ready acceptance of the modern view was found mainly in the fact of the bruit being a lengthened one when the contraction of the auricle was known to be momentary, but this is now explained on the supposition that the rhythm of the heart is really altered and that the auricle does, when hypertrophied, contract more slowly; this is now proved almost to demonstration by the tracing of the sphygmograph.

It may be in consequence of this, that the relation between the two sides of the heart is altered and, the ventricles not acting together and consequently the sigmoid valves of the two large vessels not acting synchronously, that another cardiac sound is produced constituting "the triple beat." The presystolic murmur is remarkable also for its mutability and this depends much upon the posture of the patient.

As regards the right side of the heart, I need merely mention the case of tricuspid regurgitation, met with in long standing bronchitis, where a bruit is heard over the sternum, and the fifth right costal cartilage. The veins in the neck are much enlarged and pulsate. When under these circumstances the right ventricle is gorged

and is unable to contract, the heart is said to be in a state of *asystolism.*

Of all these diseases of the valves, the obstructive forms are of less importance than the regurgitant; the aortic obstruction being of least importance, may exist for years without any accompanying symptoms; a mitral obstruction with a compensating enlargement of the auricle, may also endure for years; regurgitation through the mitral is of far more consequence, though this appears to be less important than regurgitation through the aortic orifice; a lesion not infrequently accompanied by sudden death.

The same causes which give rise to murmurs often produce a thrill which can be felt by the hand, more especially in cases of mitral contraction or stenosis. If present it assists in the diagnosis, more especially in the last mentioned condition, where being diastolic in rhythm, it can be entirely separated from the systolic impulse. Not to complicate the subject, I will not enter more fully into the subject of altered rhythm or the question of reduplication of sounds. Nor need I dwell upon the mode of production of murmurs since Dr Fagge has already explained this to you in a lecture published in the *Guy's Hospital Gazette.* He has told you how experiments have been devised for this purpose by letting water out of a vessel through openings in its sides, and that it was found that no sound was produced at these orifices by the friction of the water, but at a point beyond, where the fluid pouring out into a larger space, broke up into streamlets, and its particles jostling against one another produced the noise. In the case of narrowing of the aortic orifice, therefore, it would not be the blood passing through the chink that would produce the murmur, but the subsequent breaking up of the stream into currents as it passed into the larger vessel beyond. This fact quite accords with what many observers have stated, that bruits are heard better a little beyond the point of obstruction than at this spot itself. The experiments are full of interest, although they have not yet been made applicable to all cases of blood murmurs.

I might here allude to the *anœmic murmur* which is so commonly heard in chlorosis. It is of a soft blowing character, and limited to the region of the valves and orifices of the two large vessels. It is lost as we pass from this spot, but is heard again over the innominate, carotid and other arteries, especially if any pressure is made upon them.

The *venous hum or murmur* is best heard over the right jugular

vein in chlorotic girls; it is a continuous sound, and its intensity is much increased by the pressure of the stethoscope.

Effects on the cavities.—The next point to observe is, that owing to the changes which the cavities undergo, the difficulties arising from the impairment of the valves is, to a certain extent, compensated for, and thus the circulation through the narrowed aortic and mitral orifices being facilitated by the hypertrophied ventricle and auricle, the damaged organ may be equal to its necessities for many years, but when the ventricle gives way from being overcharged, or from degeneration of the tissue, then the failure of the organ is discoverable and the well-known cardiac symptoms ensue. It is true that the impediment to the circulation by inefficient valves will in time affect the whole system, but it is also true that it is comparatively unfelt, whilst the heart's cavities themselves remain sound. It comes, therefore, practically to this, that the symptoms are due rather to impairment in the heart cavities and tissue than to the valves; and so we shall find that our treatment is directed solely to the state of the heart itself since nothing can affect the valvular apparatus. It follows also that if the cavities were primarily affected, we should at once have the very worst forms of heart disease. This is true, and in consequence, cases where the heart has become *dilated* or *fatty* are the most formidable of cardiac affections, and they are those where very often no abnormal sounds are audible, as the valves are still intact. These degenerations may come about in many ways. With regard to the effect of violent exertion, it is still a question in the profession, but there is no question about many other causes which tend to impoverish the tissue; as age, alcoholism and obesity in general. Sometimes excessive hæmorrhage appears to have weakened the heart, and when the symptoms have been preceded by acute rheumatism, we may suppose that there may have existed an inflammation of the muscle or a myocarditis; which may have led to impairment by a fatty change or the production of a fibroid tissue pervading its structure. In cases of dilatation the closing of the mitral valve may be interfered with, and then a regurgitant bruit will exist; it will, however, in this case be a consequence and not the prime cause of the cardiac disease. If no bruit exists, it is clear that the diagnosis must be founded upon the symptoms, and we shall see that these are equally important with the local physical signs gained by the use of the stethoscope.

Effects on the other organs.—Before giving you the symptoms it

will be advisable to mention the other physical changes which the body undergoes in consequence of the impeded circulation. If the mitral valve be affected and the auricle after a time be unequal to overcoming the difficulty, the blood is thrown on the lungs, and the whole system becomes gorged, as well as all the other organs of the body. In aortic disease if the patient do not die suddenly, the same result eventually happens. First we notice the engorgement of the *lungs*, which is so great that the vessels often give way, and blood is spit up; should the patient die, large round hard masses of black blood are found effused in the tissue; this is called apoplexy of the lung. If the blood do not actually burst through the vessels, the engorgement goes on until the capillaries are completely blocked, and the alveoli of the lungs become almost obliterated by the swelling, and in consequence portions of the lung become dense and airless. We find the patient's breath become shorter and shorter every day, with indications of consolidation of the lower lobes, especially of the right side. The lung, after death, is found very dense, hard, and airless, and sinks in water. The *liver* also becomes enlarged in heart disease, and, therefore, you must not regard this enlargement as a complication, it is a necessary consequence of the engorgement. When seen on the post-mortem table, its appearance is very striking, and is known by the name of "nutmeg." The long continued engorgement of the hepatic veins causes a stagnation of hæmatine in the vessels and cells, as well as a stagnation in the biliary ducts, and this, in connection with an early fatty degeneration of the circumference of the lobules, gives with the varied colours of white, yellow, and red, the appearance of a nutmeg. The *spleen* is larger than natural and very hard. The *kidneys* are large and coarse grained. The *stomach* is often intensely injected, and its surface covered with mucus.

I need not enter here upon another class of cases where the valves are affected by endocarditis, and where the symptoms are not due so much to mechanical impediment in the circulation as to a pollution of the blood by particles of fibrin and inflammatory matter washed off from the valves. These cases of ulcerative endocarditis are fatal through the production of severe constitutional symptoms, not unlike those of pyæmia, and after death we find fibrinous masses or infarctions in the lungs, in the spleen, and in the kidneys.

THE PULSE.—Now, before proceeding further I will say a few words about the pulse. I have been questioning myself whether I

should not leave this very difficult part of my subject untouched, as it can only be learned by a practical method, and a thorough scientific attempt at its elucidation would take more time than we could allot to it. Very much has been written upon the pulse, and it forms so prominent a feature in the animal mechanism that every one thinks he has some knowledge of it; most medical men place their finger on the radial artery, and unhesitatingly pronounce an opinion upon the state of the circulation, notwithstanding that there have been those of great eminence who have maintained that it has required their life-time to find the true indications of the value of the pulse. I think such an opinion is not surprising when you consider how many different circumstances are in operation to produce the pulse and to influence it. The sphygmograph is now informing us of a number of subtle changes and conditions which the finger could not detect, and its tracings are so fine and delicate that sphygmography is a study of itself. The instrument shows that the pulse is something more than a contraction and dilatation of a vessel; the line produced by a lever placed upon it being a very complex curve. You may easily see this by making use of the sphygmometer described by Mr Wilkinson King, in 'Guy's Hospital Reports,' which is nothing more than a bristle fixed over an artery by a piece of wax; if you watch the end of the bristle you may see the vibrations which are produced. You can imagine, also, if you pressed a long piece of stick downwards on an elastic pipe through which water was being pumped, that a visible movement would be seen at its extremity. Now, if the end of the bristle just named was wetted with ink and a piece of paper passed along behind it, a series of tracings would be produced.

You may conjecture what a number of conditions are present to affect the pulse, how varied must be the tracings produced by it, and how every mark must have its corresponding cause, by considering for a moment what some of the circumstances are which are necessary for a free circulation through an ordinary vessel. Think, for a moment, of the heart as a large bag, pumping out fluid into a pipe; it is evident that the flow will be affected by the strength of the bag, by the amount of fluid it holds, and by the frequency of its contraction; it would also be affected by the state of the orifice of the pipe (corresponding to the aortic valves); and by the quality of the pipe itself, according to its rigidity, elasticity, &c., also by the freedom of the reception of the blood on the side corresponding, in the animal, to the capillary system, and even to

the state of the vessels beyond this, in the venous system; the pulse would be also affected by the quality of the fluid circulating, and again by the amount of force which was being used to keep the machinery at work—the nervous system. So you see in a simple piece of mechanism, showing the circulation of a fluid, there might be at least a dozen different circumstances present which would influence the pulse and leave their marks on the sphygmographic card.

The action of the heart itself must influence the pulse as well as the state of the orifice through which the fluid flows. It is also evident that the nature of the pipe would affect its character, and this was known to the older physicians, who regarded the blood vessel as a vital organ, and observed how its contractility was influenced by various morbid states of the system. They noticed how in inflammation, as in pneumonia, the pulse was quick and full, and at the same time, firm, owing to the increased tension of the blood-vessel, and which was also shown by the great pressure which could be put upon it without extinguishing its impulse. They noticed also how in peritonitis where the sympathetic system of nerves influenced the circulatory system, the pulse might still be hard and incompressible, whilst it was small or wiry. I must ask you to guard against confounding a hard pulse with a merely thickened vessel from hypertrophy of its coats. Moreover, it is very important to remember that in order to have a free circulation the receiving vessels must be ready to carry away all the blood transmitted to them, and so a gorged state of the capillary system will necessarily affect the pulse. You will find often in bronchitis and fevers that the blood does not flow readily through these distant vessels, the left ventricle transmits only a diminished amount of blood, and the pulse is small; you may ignorantly think in such cases because the pulse is small, that a stimulus is required to spur it on, whilst the object is to free the circulation beyond the arterial system, if this can be done. You will see by this example that the condition of the pulse points to many circumstances besides the heart which may influence it. That poverty of blood and hæmorrhage will affect the pulse, you must all have seen, and also that the state of the nervous system will influence it, as witnessed by the slow labouring pulse of cerebral disease. A number of qualities, therefore, have to be mentioned, including force, frequency, quickness, volume, as large or small, degree of compressibility hard or soft, according as the elasticity of the vessel is respectively increased or diminished.

Now if the *sphygmograph* indicate all these conditions, and even more, you may know how complex a figure must be its tracing, and how long must be the study of it before its true value and signification is recognised. The slightest changes in the conditions of the circulatory apparatus may leave their mark, and consequently minute changes, not appreciable by the finger, will be visibly drawn by this instrument. It is equally evident that this very delicacy of its action makes it quite unfit for ordinary use, and, therefore, it will probably never come into general employment. At the present time it is probable that there are not more than a dozen men in England who are competent to discuss the meaning of its figures, and amongst these we are proud to say are Mr Mahomed and Dr Galabin, two of our own men, both of whom have made original researches with the instrument. I can do no more than give you a very short and summary description of its action. Here is a tracing which is sufficient to show you that the movements of the pulse are somewhat more than a rise and a fall: you will see a perpendicular upper stroke, followed by a fall and a curve, and this immediately succeeded by another curve. Now, observers are not agreed as to the signification of these lines; the first upright one is generally supposed to be due to the sudden jerking up of the lever when the heart beats, and it is called the percussion wave or stroke; then the curve which follows corresponds to the expansion of the vessel when the blood distends it, and is called the tidal wave; this upright stroke and curve are felt as one by the finger, and are not separable; included in the pulse also, although occasionally separable, is another lesser expansion, which is shown by the second curve. It is still a question how this latter one is produced; it is believed that when it occurs the systole of the heart is over, and the aortic valves closed, and so this second curve is a kind of reaction or recoil after the blood has been sent on to be delivered to the smaller vessels and capillaries. When the vessel has lost its elasticity, and in some cases, as Dr Barlow used to teach, when there was any impediment to the circulation in front, this second wave becomes appreciable to the finger, and we have a double pulse, or as it is called a dichrotous pulse. The sphygmograph shows that dichrotism is a normal condition, although usually inappreciable to the finger.

You will remark that the first upright stroke, being merely a percussion stroke, has little to do with the strength of the pulse; this is really indicated by the tidal wave which follows, and the amount of force which can be used to affect the wave is, to a certain

extent, the measure of the tension of the vessel. I ought to say that the second or dichrotic wave has been regarded by some as corresponding to the contraction of the aorta; the first or tidal wave they have thought to be due to the systole of the heart, and the second wave to the systole of the aorta; but, as before said, the right interpretation is not yet agreed upon by all. In the well-marked dichrotic tracing, the first curve is almost wanting, the vessel is so weak that it gives way immediately under the pressure of the instrument after the first shock of blood, and then the second rebound produces a double pulse, felt by the finger. The instrument has been very useful in showing the various amounts of tension in the blood-vessels, and in the case of aneurism of the aorta, has indicated a marked peculiarity in this respect on the radial pulse.

There are many other points to be noticed about the pulse, did time admit of their illustration, as for example, what the French call "recurrence palmaire," or the rapidity with which blood returns to the distal end of the radial artery through the superficial palmar, after its compression by the finger.

Symptoms.—I have already said that when the mechanism of the heart is interfered with from imperfection of the valves, the impediment to the circulation will, in time, give rise to engorgements of the organs, with consequent symptoms, but that it is rather the weakened condition of the cavities and muscular structure dependent upon these valvular derangemeuts which produce the more marked disturbance which brings the patient under our notice. It follows from this that, if this weakened state of the heart should be the primary change, we have before us the worst form of cardiac disease. We have, therefore, two classes of symptoms and two conditions to deal with—the effects of the obstructed circulation and the weakened heart. Now, if the first has culminated in dropsy, we have remedies which, acting on the secreting organs, will relieve the loaded blood-vessels. We also have remedies which will regulate the action of the heart and increase its vigour, even before these symptoms occur.

Now, as regards the symptoms, one of the first and most important is breathlessness or *cardiac apnœa* which, as I have before described, is due to a want of relation between the heart and the lungs; the patient takes in sufficient air, but the blood is not pumped through the lungs with regularity, so that, though he

can fill his chest well with air, he is obliged to breathe frequently and irregularly or to stop for a moment before he begins to breathe again. These symptoms are quite sufficient to denote a weak heart, even if there be no bruit or derangement of rhythm.

Palpitation means that the patient is conscious of an irregular or violent action of the heart. This is due to some error in the innervation, and may be associated with a structurally healthy organ or a diseased one. In the latter case it is rarely remarked in aortic disease with enlargement of the heart, but is more usually associated with mitral disease and some ventricular dilatation; it is, however, by no means a necessary attendant on heart disease, and, as a rule, it is rather the breathlessness or the dropsy which attracts the patient's attention to his complaint than palpitation. It may be safely asserted that this is more commonly a symptom attendant upon a healthy heart than a diseased one, and that when a patient applies to a medical man on account of palpitation, the chances are largely in favour of his having no organic disease.

The patient is usually nervous, suffers from *dyspepsia*, and may be leading a life calculated to render his nervous system irritable, to say nothing of his making use of articles like tea and tobacco, which may exert a direct influence on the heart. Although it is true that palpitation is a symptom of dyspepsia and flatulence, yet it is equally true that these latter symptoms are often troublesome concomitants of heart disease. The sufferer complains, more especially upon lying down at night, of the constant eructation of gas from the stomach, and thus it is that the symptoms of heart disease are not unfrequently attributed to stomach or liver. The fact is interesting from the circumstance of the heart and stomach being supplied by the same nerve, the pneumogastric, so that when one organ is affected the other participates in the disturbance. An analogous symptom is a dry, short cough, which is much of the same nature as is observed in gastric affections.

Perhaps the most distressing symptom of all is the *sleepless night* so often met with in heart disease; as soon as the patient falls off to sleep, he starts up in fear of suffocation, and when awake is longing again for repose; in the day he waits for the night and in the night watches for the day. I have often heard patients express themselves in the words of Job, "When I say my head shall comfort me, and my couch shall ease my complaints then am I scared with dreams; so that when I lie down I say when shall I arise and the night be gone for I am full of tossings to and fro unto the dawning

of the day." These words are far more applicable to a case of heart disease than to one of rupia and periostitis as has been suggested.

All these symptoms have reference to the weak heart, but after a time the impaired circulation shows itself in *engorgements of the viscera and venous system.* The lungs are congested and increased difficulty of breathing ensues unless hæmoptysis occurs, when relief often is obtained. You will often find that after hæmorrhage from the lungs, the symptoms are alleviated, and, therefore, you must by no means necessarily wish to arrest the bleeding. In the same way the bronchial tubes become intensely congested, and a quantity of secretion is poured out from them, which also affords relief. You must not therefore say that because the patient is expectorating a quantity of mucus that he has a complication of bronchitis and attempt to arrest it. You find, on examining the abdomen, that some fluid may be present, and that the liver is considerably increased in size. This engorgement leads to some biliary obstruction, and the upper part of the patient's body may be observed to be slightly jaundiced. The legs are also œdematous. The urine is scanty and full of solid matter, being, therefore, of high specific gravity. After a time it may be found albuminous, and, perhaps, even some casts be detected; for just as in the bronchial tubes, the congestion will lead to inflammatory secretion, so in the kidneys, the congestion may pass on to an actual nephritis. This very much aggravates the symptoms, for not only does the kidney now fail to carry off the superfluous matter, but it causes an aggravation of the dropsy; and, whereas, in the first place, the anasarca occupied only the lower part of the body, we find now the arms and back of the hands becoming œdematous.

Treatment.—Now, as regards remedies, these are of two kinds; those which act directly on the heart to give it tone, and those which relieve the body of the consequences of the obstruction in the circulation. Amongst the first we have the most remarkable remedy in the Pharmacopœia; I say this advisedly, because we have no other medicine which we can hold in our hands, and give to a patient who is daily approaching his end, and at once snatch him from the jaws of death. A man may be sitting in his chair gasping for breath, and overwhelmed with the fluids of his blood collecting in all parts of his body. You give him *digitalis*, his heart may be at once steadied, and with this effect the dropsy and other symptoms

quickly depart, and before long you may actually meet him out of doors and engaged in his business. I know of several cases of this kind. Digitalis gives tone to the heart, as we know by clinical experience, as well as by observation on animals. If the chest of an animal be exposed, and digitalis administered, the organ is seen to contract more firmly, until becoming tighter and tighter, and its action slower and slower, it ceases to beat in the most complete form of systole. If the animal be previously poisoned by any substance which should relax the heart and stop its action during the time of dilatation, the effect of digitalis is even then still more striking, the drug excites it to contraction afresh, which goes on increasing in vigour until it ceases in systole as before. Now, if in disease we have exactly the counterpart of a heart in this weak state, we have also the same valuable agent at hand. Should a patient have a very irregular and weak action of the heart, digitalis is the appropriate medicine; and since this irregular action is usually a concomitant of a dilated ventricle and diseased mitral valve, it is more especially in cases where you have a systolic mitral bruit and irregular heart that you give digitalis. I could occupy you a long time in giving you illustrations of the remarkable powers of this drug. I have seen a woman pulseless with fluttering heart restored to life by its use and the withdrawal of alcoholic stimulants, and in cases where the condition of the heart above-named has been accompanied by dropsy, the latter also has subsided as the organ has regained its tone. When the patient simply has this troublesome affection of the heart without further symptoms, I order ten drops of the tincture three times a day for a few days, and watch its action. If I was not seeing the patient for some time, I might order half the dose.

Another tonic for a weak heart is *iron*, and I therefore often combine the two remedies, digitalis and iron. Many persons have had their lives prolonged for years by the use of these two remedies. When the digitalis has been given sufficiently long to produce the required result, you may give the iron alone. Or you may join ammonia with iron. A simpler medicine is ammonia, with a bitter like senega, and with these we often combine henbane, on the supposition that it has a quieting influence on the heart, You will see from these observations that digitalis can have little effect in the case of the quiet and regular acting heart, such as is met with in aortic disease, and as a clinical fact, we find this medicine of much less value in these cases. Digitalis is a diuretic, and is therefore useful

in all cases where there is dropsy. Here we wish to act on the secreting organs to free the body from the dropsical fluids and for this purpose there has been for many years past a pill in almost universal use composed of squill, digitalis, and mercury — for example, a grain of each of these (using the pill hydrarg.) three times a day. The effect is not only to quiet the heart, but to increase the action of the kidney and liver, and so relieve the patient of the fluid. At the same time a purge is of eminent service, and patients often express themselves more relieved by a jalap powder two or three times a week than by any other remedy. You know that we have been using of late the resin of copaiba also as a valuable diuretic medicine: for sleepless nights we are obliged to give opiates as a quarter of a grain of morphia with ether as a draught, and find the remedy highly beneficial. When all medicines fail and the obstruction to the circulation is very great, we are forced to prick the legs, so as to allow the fluid to drain away; since it will not pass round in the course of the circulation, the only resource we have is to let it off. The practice is not unfrequently followed by dangerous erysipelatous inflammation, but at the same time the patient's life may be prolonged for weeks or months by its judicious use.

ANEURISM OF THE AORTA

"An aneurism is a circumscribed tumour full of fluid or solid blood communicating directly with the canal of an artery and limited by the membrane called the sac." There was at one time a controversy as to whether the term aneurism could be strictly applied to the case of dilatation of a blood-vessel and whether it should not be limited to saccular pouches formed in a particular way, and also whether special names should not be given according to the number of the arterial coats of which these consisted. Divisions of this kind, however important from a pathological point of view, are of no value clinically, seeing that the same aneurism may alter its characters as it progresses in age and size. It might, for example, commence as a sac composed of all the arterial coats, but subsequently as the internal and middle coats by turns give way, be left with no covering but the cellular tunic. There is also a division of aneurisms according to their shape, but this again need have no mention in this place. One reason for the amount of attention which the different forms of aneurism received and their separation from dilatations of arteries was

owing to the fact that there is a greater proneness to coagulation in a pouch which is circumscribed and of which the coats are diseased. We now call every case a "true aneurism" which answers to the definition above given, a "false aneurism" being the case of effused blood communicating with an artery, where there is no limiting membrane.

In the case of the aorta with which we have alone to deal, it is as well to make a distinction between an aneurism and a dilatation although the two conditions are so constantly associated that we are often forced to adopt the expression aneurismal dilatation. In the case of a small artery the distinction would not be important but it is far different with the aorta which has to transmit the whole current of blood immediately it leaves the heart. A circumscribed aneurism for instance coming off as a pouch from the aorta might in no way interfere with the circulation, whereas a dilatation of the whole vessel might seriously involve the valves or from a loss of its own natural elastic properties set up all the phenomena of cardiac disease. You can easily see how a thickening of the part constituting the sinuses of Valsalva would prevent the due closure of the valves or if the arch itself was converted into an unyielding, inert bag, it would interrupt the whole circulation of the blood.

Aneurisms of the aorta however are mostly circumscribed and therefore in no way interfere with the circulation of the blood through the vessel, the symptoms being due entirely to their mechanical pressure on adjoining organs. The ordinary characters of a superficial aneurism are therefore wanting, as those in the aorta are rarely palpable. In considering therefore what are the symptoms of the disease we must inquire how the parts with which it is related may be affected by its pressure rather than by reflecting upon the properties of an aneurism: we shall now see why it is often difficult to diagnose between cancer of the chest and aneurism seeing that they are both acting injuriously in the same way, simply as tumours.

You will perceive too that as the seat of aneurism may vary so the implication of particular parts must vary and the corresponding symptoms differ considerably; consequently in no two cases are they exactly alike. If you think for a moment of the close packing together of the aorta, trachea and bronchi, œsophagus, pneumogastric, laryngeal and sympathetic nerves you will see how impossible it is for an aneurismal tumour to grow in this region without interfering with some of these important structures.

Now, in the first place, let us take the case of aneurism of the *ascending aorta*, and you will see from its position how few may be the symptoms attendant on its formation and growth. It begins as a small pouch, and as it grows it presses forward against the sternum and costal cartilages on the right side until these are absorbed, and it appears as a pulsating tumour on the surface. So few are the symptoms sometimes attendant on its progress, that the patient may have felt nothing but a little pain until he perceives the external swelling by his eye or hand.

When you examine a patient affected in this way, you may, perhaps, detect a pulsation or a thrill when the hand is laid on the chest, but you cannot see the pulsation. If this is the case you should place your eye on the same plane as the man's chest either by stooping as he lies on his bed or by looking over his shoulder from behind, when he is in the erect posture; in this manner you may observe a pulsation which was not visible on direct vision. You should also bear in mind (which is very important in the diagnosis of all aneurisms) that there is an expansion as well as a throbbing of the tumour. When, for example, you place your hand on an aneurism, it is not only raised by the impulse, but at the same time it becomes cognisant of a feeling of expansion. This may be shown on an aneurism of the chest by covering it with a piece of plaster in whose edge a slit has been made; when the swelling dilates this slit or notch in the plaster is visibly widened. On placing the ear over it a bruit may possibly be heard; if so it would be systolic in rhythm, and caused by the blood rushing through a narrow opening into a large sac, or by its passing over a roughened and diseased aorta.

An aneurism of this kind may eventually form a very large tumour in front of the chest, and the skin becoming implicated there may be constant fear of its bursting. Such an event, however, is very rare. I have never seen an aneurism of the aorta rupture externally, although I believe cases of the kind are recorded. They prove fatal by interfering with the circulation or by bursting into the pleura or pericardium; when into the latter, death is not caused by loss of blood but by strangulating the heart and so arresting its action.

In aneurism of the arch of the aorta the symptoms vary with its site, as the parts which are liable to be compressed are different in every case; therefore, before you can understand the symptoms we must first see how they may be produced.

I have seen an aneurism press upon the *pulmonary artery* and in this way set up a dropsy and all the symptoms of cardiac disease, and I have more than once known the sac burst into this vessel. Pressure upon the *trachea* and *bronchi* is very frequent; in consequence of this there may be great difficulty of breathing or sense of suffocation, and as the tube becomes implicated in the tumour an inflammation and ulceration is set up, accompanied by mucous secretion tinged with blood, and perhaps, finally, a fatal hæmorrhage takes place. In this case, although there is difficult and perhaps wheezy respiration, the voice is unaffected, showing that the larynx is not implicated. If one bronchus is compressed we may hear the air passing through it with a noise and the lung on that side imperfectly filled.

The *œsophagus* may be pressed upon producing dysphagia, whereby the patient is obliged to swallow twice before the food will pass. The aneurism may eventually break into the tube and the patient quickly die after bringing up a large quantity of blood; or without bringing it up, the same result may occur by its passing into the stomach or bowels.

The *recurrent laryngeal nerve* may be compressed giving rise to some of the most remarkable and characteristic symptoms of aneurism. The left is the one most usually affected as it passes under the arch of the aorta. You know that in the respiratory process the larynx is actively opened by its muscles through their appropriate nerve, and that the organ is also under the control of the will during speaking. The recurrent or inferior laryngeal nerve contains the fibres both for respiration and speech, consequently, if this nerve be compressed, both these functions are interfered with, and the patient can neither breathe well nor speak well. He breathes with some difficulty and with a peculiar hoarse, crowing, or stridulous noise; it is this sound which strikes the ear of the medical man as suggestive of paralysis of the vocal cords, and, therefore, some implication of the recurrent laryngeal nerve. When examined by the laryngoscope it is found that one vocal cord is paralysed and immoveable. If both nerves are compressed the glottis would close and death would inevitably ensue; but it is remarkable also that this closure and actual suffocation will occur when only one nerve is affected. It has, therefore, often happened that tracheotomy has been performed and with temporary success. Why complete paralysis should occur under these circumstances is not very evident, but it has been suggested that disease of the

nerve may continue upwards until it reaches the centre where it meets its fellow, and this it may implicate in its malady. It may, however, be remembered that in the case of aphasia it has been suggested, in order to account for the phenomena observed in this condition, that the larynx may be stimulated to action by the nerve on one side only. After death the nerve is found flattened and the muscles of the larynx atrophied. I might mention that in the case of roaring horses the muscles of one side are wasted owing to atrophy of the laryngeal nerve; the cause of the atrophy being due to enlargement of the glands and other unknown causes.

Nerves of the pulmonary plexus as well as the pneumogastric itself may be involved in the aneurism, and as a consequence a pneumonia is set up which not unfrequently brings the case to a final issue. Whether the inflammation is caused directly or indirectly by implication of the nerves is a question which has its counterpart in cases of nerve destruction in other parts of the body; whether, for example, the nutrition of the lung is directly affected, or whether a paralysis of the bronchial tubes causes a collection of secretion to take place, and so the pneumonia is gradually set up. It is remarkable that exactly the same thing occurs with carcinoma of the œsophagus and for a similar reason.

Pressure on the *sympathetic in the neck* is also productive of another phenomenon which has been repeatedly noticed, contraction of the pupil on that side. Other tumours besides aneurismal would of course do the same.

Alteration of the pulse in many ways is also constantly observable in aneurism of the aorta. If the subclavian on either side is involved, it may sometimes be found retarded and the pulse at the wrist be slightly behind that of the other. I might also mention that in various kinds of aneurism of the aorta the pulsation in the tibial arteries is often more delayed than in health. The most striking peculiarity, however, in the wrist pulse is the inequality, one being markedly smaller and weaker than the other. This may be due to a simple mechanical cause, as from the partial closure of the subclavian artery at its origin in the aneurismal aorta or from actual pressure on the vessel by the aneurism itself, but the inequality most usually occurs in another way. The subclavian arising from the aneurismal pouch has not the same pressure exerted on it as when forming a branch of the natural elastic aorta, for the blood passing from the heart into a large inelastic sac loses the power of propulsion which it otherwise would have had exerted upon it.

The sphygmograph generally demonstrates this in a most marked manner, and showing the value of this instrument, it has been able to prove a want of blood pressure on both sides, and the implication in the aneurismal lesion of the innominate artery as well as left subclavian, at a time when the finger could perceive no great peculiarity, or at least no difference in the pulses of the two sides.

I might here say that if the aneurism does not interfere with the action of the valves, the heart is not altered in size; there being no necessary hypertrophy in this disease.

Symptoms.—Having now heard what morbid conditions are liable to occur you will be prepared for the corresponding symptoms. These, of course, may be of the most varied character. Probably the patient, who is a middle-aged man, comes to you complaining of pain in the chest and some difficulty of breathing increased on exertion, or he may have a peculiar stridulous respiration or ringing brassy cough which may excite your suspicion. These symptoms are of the vaguest character, but of a kind which ought to suggest to you the possibility of aneurism, and of course you look for it; on doing so you might find pulsation in one part of the chest, and on auscultation a bruit and, as is often the case, the second sound of the heart accentuated; you might also discover evidence of pressure on a bronchial tube by imperfect respiration on one side, and if the voice were stridulous you would perceive paralysis of the vocal cords on examination by the laryngoscope. If the trachea were actually involved in the aneurism you might also have expectoration of mucus and possibly blood; on inquiry you might also learn that the patient had some difficulty in swallowing his food. The symptom of course would vary with the situation and size of the aneurism. If any portion of the ascending aorta were involved a pulsation might exist on the right of the sternum, and if the lower part of the arch were affected it is possible to have pulsation on the left side; in each position there might be a bruit. It is not unusual to find a bruit at the origin of the aorta over the valves, but, if this is diminished in intensity as you pass your stethoscope across the sternum and then again becomes intensified over the second right costal cartilage, you may infer that there is a source for the murmur in the vessel itself, and if it is also very distinct over the first rib that the innominate is probably also involved. Occasionally but rarely a diastolic murmur may exist and it is not uncommon to feel a thrill when the hand is placed over the aneurism.

Amongst the more complex cases we have that of aneurismal dila-

tation; where the whole arch is much deformed, the walls thickened in some parts, and attenuated in others, so that distinct pouches are formed, and at the same time the internal surface is scabrous or covered with cretaceous plates. The symptoms are due in part to the inefficient action of the aorta in driving the blood, and also to the local influence of the several pouches on the parts with which they come in contact.

In *aneurism of the descending part of the arch*, the left bronchus and lung may be more especially involved, or the vertebræ may be eroded by the pressure exerted upon them, causing intense pain by implication of the intercostal nerves. The disease may be fatal by a rupture of the aneurism into the pleural cavity.

In *aneurism of the thoracic aorta*, the earliest symptoms are those of pain, the distension of the vessel itself will produce it, but it is not so great as when the intercostal nerves are involved in the disease.

Abdominal aneurisms may have various seats as immediately beneath the diaphragm and in its course below; they may occur also in the cæliac axis and more rarely in the renal and mesenteric arteries. These are more readily diagnosed than thoracic aneurisms, for if they are of any size they may be grasped by the hand and their pulsation and expansion easily felt.

Treatment.—There is a possibility of cure of aortic aneurisms, since they are occasionally found on the post-mortem forming solid tumours from solidification of the blood within them. The object being to cause a deposition of fibrin in the sac, we adopt that plan of treatment which will most readily tend to promote it. This is accomplished by retarding the circulation, acting on the sac in various ways to facilitate the precipitation of the fibrin and by doing all we can to produce an excess of this substance in the blood. In abdominal aneurisms which can be manipulated like those of the extremities local measures may be adopted, and several cases have now been recorded where a cure has been effected by pressure above the sac. Where this cannot be done as in thoracic aneurisms the only means feasible are those which may tend to increase the amount of fibrin and facilitate its deposition in the diseased parts. With this object various medicines have been tried, as digitalis, antimony, acetate of lead, but all without result. The only method which has ever been attended with success is that proposed more than a century ago by Valsalva, and that is by almost starving the patient whereby the circulation becomes so feeble and the blood so thick that it readily coagulates. It is admitted that the plan is rational since in starva-

tion the amount of fibrin is in excess and because a certain number of successful cases have been recorded to warrant the practice which Valsalva proposed. He recommended that the patient should be bled several times for some days, and that nothing should be given him but a little water, and in this way should be brought down to death's door; in this dead and alive condition the blood would coagulate in the aneurismal sac and the disease be cured. The patient was then again restored by a gradual return to good living.

Dissecting aneurism.—This is the name given to that disease where the internal coat of the aorta undergoes a laceration, and the blood passing through the fissure, extravasates beneath it, gradually tearing away one coat from another until the internal one is completely separated from the others, or rather it is that the middle coat itself becomes torn throughout the whole of its circumference and perhaps for a great part of its length, so that blood becoming extravasated in the arch of the aorta may extend downwards as low as the iliacs. The laceration is generally attended by a sudden intense pain and perhaps with syncope, after this a reaction may take place followed in a few hours by a further rupture through the external coat with speedy death.

ANGINA PECTORIS

This is a nervous affection of the heart occurring for the most part in men above middle age and very rarely in women. As a rule the valvular apparatus is healthy, but the muscular tissue is fatty and the coronary arteries ossified. One of the best specimens of this condition of the heart in the museum came from a patient of mine who committed suicide during an attact of angina. The degeneration of the muscular tissue probably implicates the nervous supply of the heart, and so causes the spasm and the accompanying agony. The sufferer generally has a quiet acting heart affording nothing abnormal to the stethoscope, but he is breathless on exertion which is sufficient to indicate a weak condition of his heart. On an extra effort, more especially in walking up a hill, a paroxysm comes on, the patient immediately stops, for he feels that if he moved a step further he would instantly die; the respiration is not affected although the sufferer often holds his breath as he thereby gains relief, the pulse if felt is steady and regular as before. These cases are usually treated by antispasmodics but with little avail, and of late years the nitrite of amyl has come into use for inhalation. The only medicine I have seen do any good is arsenic long continued.

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